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ACUTE HEMATOGENOUS OSTEOMYELITIS*

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THIS paper constitutes a preliminary report, its object being to present in a concise manner our present conception of the pathology, pathogenesis, and treatment of this disease, with reference to the recent literature, with brief consideration of cases which have come under our observation, and with certain recommendations for its treatment.

The condition is the result of a local settling of bacteria usually in the metaphysis of a long bone, and predicates a preëxistent bacteremia, which may, however, be subclinical, without signs or symptoms.

It involves almost exclusively the long bones of the upper and lower extremities; hematogenous infection of flat and irregular bones such as the ilium[27], jaws[14, 23], patella[7], scapula[8], skull[24], and vertebrae[12, 21], is certainly seen, from time to time, but does not enter into this discussion. The usual order of frequency of involvement, according to Beekman[4], is tibia, femur, humerus, fibula, radius, and ulna. Pyrah and Pain[16] list the frequency as follows: (1) upper tibia, (2) lower femur, (3) lower tibia, (4) lower fibula, (5) lower radius; thus it is seen that the greatest number of cases are in proximity to the knee joint. It is a disease primarily of childhood and adolescence, the period of active growth, most common between the ages of five and fifteen, and more frequent in boys than girls.

For a proper conception of the pathological process which takes place, a knowledge of the anatomy and physiology of bone, particularly in childhood, is indispensable, and a brief exposition of them is herewith given. The long bone has a shaft, or diaphysis, and at each end an epiphysis; the two are separated by an epiphyseal, or conjugal cartilage. The diaphysis, except at its ends, is hollow, containing the medulla; at each extremity the bone is cancellous; one might say semisolid or porous, and this part, the one most frequently involved in osteomyelitis, is called the metaphysis. The epiphyseal, or conjugal, cartilage, separating the metaphysis from the epiphysis, contains active cells which produce bone and are responsible for longitudinal growth. Closely encasing the bone is the periosteum, in two layers, the outer or fibrous one, and the inner or osteogenetic layer, which produces the concentric growth of the bone. The periosteum is continuous with the epiphyseal cartilage; in bones which have many muscular attachments like the femur, it is with difficulty detached, but in others, like the tibia, it is easily stripped up. Infectious trauma, if it destroys the epiphyseal cartilage, may result in cessation of bone growth; if, on the other hand, its action is one of irritation and stimulation of the cartilage, overgrowth and abnormal length may occur.

The circulation to the diaphysis itself is twofold, through the nutrient artery and its branches, and through the small periosteal vessels. The nutrient artery enters the shaft somewhere near its middle, divides into ascending and descending branches, and supplies the medulla, inner parts of the shaft, and the metaphysis. In the metaphysis the vessels form terminal venous loops where the circulation is slower than elsewhere[20]. The outer portions of the shaft and the metaphysis receive blood vessels from the periosteum; in the diaphysis itself the branches from the central nutrient artery anastomose with those from the periosteum; in the metaphysis the situation is not so clear; it used to be thought that there was no anastomosis between the nutrient and the periosteal vessels, but now we believe that there probably is such a communication. The epiphysis receives its blood supply from both the periosteal and the capsular vessels. Blood vessels do not traverse the epiphyseal cartilage in either direction.

The organism most commonly present is the staphylococcus, next the streptococcus; much less frequently one finds others such as the bacteria of influenza and pneumonia, and very rarely the typhoid bacillus. In one hundred cases studied by Albee[1] there was a pure culture of staphylococcus in forty per cent, streptococcus in fifteen per cent, and in a substantial proportion a mixture of the two. There can usually be found somewhere in the body a preëxistent primary focus of infection, such as furuncle or abscess. From this focus, bacteria have been set

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free into the circulation, and have gone to ground, so to speak, in the terminal venous loops in the metaphysis. It has been generally accepted that antecedent local trauma, creating a "locus minoris resistentiae", has in most cases occurred. A history of such an event can often be obtained, and the fact that the disease is most common in active boys constitutes supporting evidence. On the other hand, in a long review by Reischauer[18], in 1932, evidence is adduced which leads the author to doubt that there is any noteworthy etiological connection between trauma and either osteomyelitis or bone tuberculosis.

When the invading bacteria come to rest in one of the terminal vessels of the metaphysis, they begin to multiply and an immediate localized infectious process is set up, with the accompanying symptoms and signs of pain, localized tenderness, limitation of motion, and high temperature, with its concomitant subjective manifestations. Pus accumulates, and the contiguous vessels become thrombosed, with resulting interference with circulation. In the typical case the pus burrows toward the epiphyseal cartilage, thence on along the epiphyseal line to the surface of the bone; from here, if the epiphysis is extracapsular, it burrows under the periosteum, stripping it up; if the epiphysis is intracapsular, it may rupture into the joint, but this event is uncommon[5, 25]. The tenderness over the bone appears very early, but swelling and edema over the involved part are late occurrences, and are evidences of an advanced process. Sometimes the pus burrows back into the medullary cavity, and, as this happens, more blood vessels are destroyed, and there is still further interference with the cortical blood supply. Furthermore, when the pus has burrowed under and elevated the periosteum, the little periosteal vessels which supply the outer surface of the bone are also damaged. Interference with the circulation results in the local death of bone, forming the sequestrum (or sequestra) which may thus result from thromboses within the metaphyseal, in the periosteal, or in the intramedullary arterioles. The sequestrum is a foreign body, detached slowly from the healthy bone, and though it may be in part absorbed, it usually has to be cast off or mechanically removed. As the sequestrum develops, the periosteum or endosteum forms new bone which gradually gets thicker and stronger and constitutes the involucrum.

The bacteremia which precedes the osteomyelitis is usually unrecognized and, as noted above, subclinical, but with the onset of the acute bone process there may develop a fulminating septicemia with its typical clinical picture, and often with subsequent involvement of other bones, constituting what is in reality a pyemia. In these cases the local osteomyelitis, while demanding appropriate treatment, is often secondary in clinical importance to the virulent blood-stream infection, which may often in itself be fatal.

In some instances the infection becomes localized in the metaphysis, forming a small low-grade chronic collection of pus, called Brodie's abscess. This occurs in most cases in the tibia, and the other bones usually involved are, in order, the femur, humerus, radius, and ulna[17].

The death rate in acute osteomyelitis, as given by numerous authors, varies from 13 to 34 per cent. In a series of ninety-eight children, carefully studied by Beekman[4], the mortality was eighteen per cent and in the ninety-four cases reported by Ingelrans it was 15.95 per cent. The cause of death is usually septicemia, though occasionally listed as pneumonia, or some other process which is secondary to the original acute infection. The untoward and late results, deformities, disabilities and recurrences, will be discussed in a subsequent communication. The early and correct diagnosis of acute osteomyelitis is of untold importance, and on these depend proper treatment, avoidance of long illness, recurrence, deformity, loss of wage-earning ability, and life itself. In brief, any patient, especially between the ages of five and fifteen, whether previously in good health or not, with a sudden onset of pain and localized tenderness over a long bone, near a joint, with elevated temperature and elevated white blood cell count, and the usual symptoms of acute infection, must be considered as a case of acute osteomyelitis. Scurvy usually occurs in younger children, or rather infants, and is now rare; tuberculosis is slower in onset and is ordinarily a joint infection; acute septic arthritis presents typical signs of joint involvement and, of course, fluid in the joint cavity. It is true, as noted above, that where epiphyseal cartilage is intracapsular there may occur a pyogenic arthritis as a result of the traveling of pus from the metaphysis via the epiphyseal line into the joint, but even this will be preceded by typical signs of a localized metaphyseal infection. The x-ray will not show any evidence of osteomyelitis until elevated periosteum has developed new bone containing calcium, or until bone absorption has occurred, and this will not be for two or three weeks; to postpone diagnostic decision until this picture has developed is fatal. Infinitely more harm has been done by ill-advised procrastination than by early, even mistaken, radical exploration of the bone.

For proper treatment the "sine qua non" is, as I have said, early diagnosis. The whole secret, the proper prevention of unfortunate sequelae, is the institution of early and adequate drainage of the infected area in the bone. The diagnosis must be made on the strength of the fever, pain, local tenderness, white cell count, and general constitutional picture. Local swelling, edema and redness, and changes in the x-ray

findings, must not be waited for. It is not particularly harmful to trephine the bone in the case of mistaken diagnosis for the wound will heal promptly; on the other hand, procrastination will allow the process to get out of hand and develop into an extensive and chronic disease.

Under ether anesthesia an incision is made over the area of most marked tenderness, the bone laid bare, and if there are no local signs of edema, or pus formation, a hole drilled into the bone with a quarter inch burr; this is repeated several times until pus is found and an adequate opening for drainage has been made. It is not sufficient, if a subperiosteal abscess is found, simply to drain it, as some have recommended, but the bone must be widely laid open. The epiphyseal cartilage, the epiphysis itself, and of course the joint cavity, must be scrupulously avoided. When the abscess in the bone has been located, and a sufficient opening produced, the best treatment is, at the moment, to pack in some gauze impregnated with vaseline or boric acid ointment, and to change this in a few days, inserting tubes for irrigation. Frequent dressings are necessary, and may at first require an anesthetic. We believe that frequent washing of the wound with Dakin's, or some similar solution, is preferable to other methods of treatment. By renewed packings, by frequent irrigations, great care, and rigid asepsis (that is, care to avoid contaminating the already present sepsis) the wound should heal in a comparatively short while. In this acute stage the one important thing is careful drainage, and the continuance of drainage, and infinite pains to avoid undue injury to either periosteum or bone.

At the first operation, in the very acute stage, no sequestrum will be found. The actual task is simple; the only difficulty being, at times, that of finding the pus. When the case has been neglected, and comes in after an illness of two or three weeks or more, the picture will be very different, with advanced local disease, and often complicated by both a general septicemia and metastatic foci of osteomyelitis. The area of early bone destruction, resulting from thrombosis of a terminal metaphyseal vessel, is easy to comprehend, though hard to visualize; when there are, in addition, thromboses of vessels in the periosteum, in the medulla, and the cortex below the metaphysis, the whole situation is locally complicated and confusing, and the bone necrosis is irregular and unpredictable. Even the careful scrutiny of an x-ray film will often fail to tell one the exact extent of bone destruction, and the precise limits of sequestrum formation.

In the neglected acute cases, those which have been allowed to progress from two to four weeks, the operative procedure must be a judicious combination of conservatism and radicalism, with a leaning toward the former. Too eager removal of bone, especially if the epiphyseal cartilage be damaged, may be productive of lasting harm.

The treatment of chronic osteomyelitis, its indications, and the technical points of operation and after-care, are beyond the scope of this paper. It is our opinion that the various forms of therapy most discussed at the present time[9, 10] the bacteriophage, the use of the blow-fly maggots[6, 22] and the Orr treatment[13, 15] are laudable attempts which do not solve the problem. They all have points in their favor, and each may be best in a certain case, but the very fact that there are so many different recommended forms of treatment is evidence that no one is ideal, and that the answer has not yet been found. Or, at least, the answer has not been found to the cure of neglected and chronic cases; the solution lies in educating the public to the great importance and danger of acute pain, with fever, in the bone of an extremity, and in educating the profession to the necessity of immediate exploration of such a case.

At the Massachusetts General Hospital there were, in the ten years from 1923 to 1932, inclusive, 1046 admissions for osteomyelitis of all kinds, with the following distribution:

Femur	239	22.8%
Tibia	155	14.8%
Jaw	146	13.9%
Humerus	82	7.8%
Fingers	67	6.4%
Toes	40	3.8%
All others	317	30.3%

The mortality was 43, or 4.1 per cent. The fact that this figure is so low is because there are included many cases of minor osteomyelitis, not the acute hematogenous and fulminating ones. A substantial number of them were multiple, and reëntries were frequent.

These entries into the Massachusetts General Hospital, over 1000 in ten years, represent 100 per year, or roughly two each week. As this Hospital is only one of many in the city, the total admissions in Boston alone can safely be estimated at several hundred a year, and the incidence in the country at large would be many thousands a year. Each case represents a material loss of time and money, with a variable degree of crippling, and all together a noteworthy loss of life.

Impressed by the great problem here presented, and with the purpose of arriving at more successful methods of handling the cases, we have formed at our Hospital an Osteomyelitis Clinic, or Service. At the head of this are one general surgeon and one orthopedic surgeon, each with assistants. The ambulatory cases are seen on one specified day each week in the Outpatient Department, and when the number of patients increases, as for a time it will, two days

will be set aside for this purpose. Regular ward-rounds, once a week, for the discussion of especial problems, are being instituted. The success of this clinic, its difficulties, and its findings, will be reported to you later.

At the outset we urge that this disease, a cause of so much economic loss, suffering, and even mortality, be attacked in the stages before irremediable damage has set in, when the diagnosis is perhaps more in the realm of art than that of science, and when cure is possible. Instruct the laity that acute unaccountable pain in an extremity is just as serious as pain in the abdomen, and requires immediate medical or surgical attention. Teach the young surgeon that procrastination does infinitely more harm than exploration, even if, at times, the latter be fruitless. When a true comprehension of the importance of the situation is attained, then, and then only, will osteomyelitis be brought under satisfactory control.

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DISCUSSION

DR. ROBERT B. OSGOOD, Boston, Mass.: The members of the New England Surgical Society should be grateful to Dr. Miller for directing their attention to this type of acute bone disease because sins of both omission and commission are being committed in its treatment. With almost all he says I am in hearty accord. I believe that his statement that the staphylococcus is the organism usually to be held responsible needs qualification. My impression, confirmed by a recent analysis of the Boston Children's Hospital cases made by Dr. William Green and Dr. James Shannon, as yet unpublished, suggests that the streptococcus, usually hemolyticus, is recovered twice as often as the staphylococcus in children under two years of age. The disease is not rare in this age group.

This knowledge has a bearing on treatment which seems to me important. Streptococcus lesions in infants require less complete drainage. High temperature is common, but by no means an unfailing sign. The reaction in the early stage may be very slight in very young children.

Dr. Miller calls attention to the pyogenic arthritis which may ensue in an intracapsular lesion. It is well to remember that heat, tenderness, and synovial effusion may be noted in joints contiguous to an extracapsular lesion and may demand no treatment other than drainage of the metaphyseal focus of infection.

In children I am not in entire agreement with Dr. Miller that as soon as the diagnosis is *clear*, immediate operation is always indicated. We shall all encounter cases where the diagnosis to us is all too clear, but has not been to our colleagues by whom the cases have been referred. The lesion has existed for some days or even weeks, and the child is dehydrated and very ill. Twelve or even twenty-four hours under sedatives and glucose and salt solution intravenously, per rectum, or subcutaneously, will offer a far better prognosis for whatever operative procedure is indicated.

On theoretical, and I believe also on practical grounds, I dislike searching for pus in the metaphysis by repeated drill holes into the bone. If pus is not found, new avenues of infection may have been opened into bone previously uninfected. Strong, direct light is necessary. I have personally used a head mirror. It seems to me more surgically intelligent to remove a generous cap of cortex over the metaphysis, and with sight and a small probe or tiny curette to gently search beneath for the focus, striving not to break through nature's attempt to wall off the infection. In this attempt she is often quite successful, as in a Brodie's abscess, which at its inception is often an acute osteomyelitis, and we should strive to preserve rather than to break down nature's protective barriers. Drainage may then be instituted more completely and safely when the location and extent of the focus is known.

I could tell you stories illustrating the points I am trying to make. I entertain the possibility of many osteomyelites curing themselves. Please do not understand me as believing that we should not drain the lesion as soon as we are sure of its existence, if the condition of the patient does not contraindicate immediate operation. I believe we should, but in osteomyelitis in infants under two years of age, I have seen a number of cases that have cured themselves, coming on as multiple lesions.

In children I must speak a word in favor of infrequent dressings and the so-called Orr method. I think I may say that I have observed no disaster to follow its administration in children in a considerable number of cases. During several years' trial

at the Children's Hospital, I am sure flatter charts have been the rule. Anesthetics are not good for sick children, and fear inhibits the capillary flow. Dr. Ober has said that temperature rises will tell the dates of dressings, without the nurse's notes.

I am in hearty accord with what Dr. Miller says in regard to surgical conservatism in old and neglected cases. A serial set of lantern slides may help to sharpen the point he has made. I know I may speak for the Society in thanking Dr. Miller for his most timely and helpful paper.

DR. H. BERTRAM LAMBERT, Bridgeport, Conn.: It is perfectly obvious that it is almost impossible to add to Dr. Miller's able paper with Dr. Osgood's discussion, but there are one or two points I should like to emphasize, if I may, particularly that with the rather alarming prevalence of acute osteomyelitis, it ceases to be entirely a problem that should be impressed on the general practitioner to not wait for an x-ray and not wait for a swelling.

I think if you will mull over in your own minds a large number of these cases, you will find that the general practitioner has excused himself by saying that the x-ray has shown nothing and there has been no swelling.

The question of trauma I think is quite a problem for most people who have not read recent literature to meet. All the recent literature on this subject points without question to the infectious origin with trauma entering as a doubtful etiologic factor.

I have seen several cases of scarlet fever in which there was no complication of otitis media that could be noticed. They were rather interesting and developed quite early in the disease.

I noticed in reviewing the literature in one case that was badly missed, the lesion was in the upper end of the humerus, the swelling occurring at the lower end of the humerus, and the case was operated on at the lower end rather than the upper end, which emphasizes a very important point which is that the pain of this condition is almost a fingerpoint pain; it isn't diffuse. It occurs over the beginning of the lesion if one sees the case early.

DR. LYMAN ALLEN, Burlington, Vt.: I rather hesitate to say anything about osteomyelitis in the face of the people who have had so much more experience than I have had; I agree with what Dr. Miller has said in every detail, I think, except when he discusses the question of frequent dressings after operations for early acute osteomyelitis. I do not agree with that. I believe that the periosteum should be split, the cortex opened, and the little pus cavity gently cleaned out without opening into the marrow cavity. Then this little abscess should be packed with vaseline gauze and left alone (the Orr treatment) for days and weeks. The patient treated thus will get well and is going to have much less physical and mental upset caused by pain, fear, and dread. I do not think we have any reason for syringing out the cavity. I am speaking only of cases seen and operated upon early.

With that exception I should like to say I appreciate Dr. Miller's paper very much, but I do know from personal experience that frequent dressings in the early, young cases of osteomyelitis, are not necessary and I believe they are harmful.

DR. WILLIAM E. LADD, Boston, Mass.: I hope the Society will not go away with the impression from Dr. Miller's paper that the operation on acute osteomyelitis is necessarily an emergency operation. I believe the surgical principle of selecting the time for the operation is quite important. If a child comes in with a temperature of 104° or 105°, from an acute fulminating osteomyelitis, is subjected to an immediate operation, the mortality will be unduly high.

I believe it is very important to get the patient in as good condition as possible before opening the bone.

Now, lest you interpret that to mean that I would go to the other extreme and have the Society get the impression from Dr. Osgood that we don't want to operate on them at all, I wish to say I think that would be equally harmful. I think it is a matter of judgment and very important judgment, to select the appropriate time for operation on these acutely ill patients, I believe twenty-four, forty-eight hours, or even two or three days spent in preparing the patient for operation, is often time very well spent.

One other point: on the small infants, the children under two, I think the mortality will be lowered by not being very radical. It is our practice at the Children's Hospital, to open the periosteum only at the first operation on the small infants. They get very much more adequate drainage, at the epiphyseal junction, than the older children or the adults. The lesser operative procedure is attended by a much lower mortality than the more radical one on the small infants.

DR. RICHARD H. MILLER, Boston, Mass.: I really did not mean to convey the impression that I thought that operation in every acute case must be done, within six hours, and I quite agree with Dr. Osgood and Dr. Ladd that there are many instances where one must treat (as Dr. Osgood says) the patient for a while until the latter's condition has been brought up to such a state that surgical operation is safe and feasible.

In regard to occasional cases recovering spontaneously, I think there is no question that once in a great while this happens. It was impressed on me only two days ago when I was asked to give my opinion about a case where a very acute process had apparently started at the lower end of the tibia, and then it subsided before the local physician had been able to make up his mind what was going on, and I think it was an abortive, acute osteomyelitis which took care of itself.

I wish to congratulate Dr. Osgood on his splendid judgment in that case in which he left the sequestra in place.

DR. OSGOOD: It wasn't mine, no credit to me.

DR. MILLER: Just one last word. I do think that in children one can't do frequent dressings the way one does in adults, and certainly there are instances where a modification of the technique has to be employed.

DIABETIC COMA*

BY ALEXANDER MARBLE, M.D.,† HOWARD F. ROOT, M.D.,† AND PRISCILLA WHITE, M.D.†

ALTHOUGH everyone agrees that few should die from diabetic coma and that none should develop it, we have had 55 cases since our last report[1]. In the last ten months there has been only one death among 32 consecutive cases treated at the New England Deaconess Hospital. This death was in a patient (2786) who, although rescued from diabetic coma, died from an accompanying pneumonia of sudden onset and rapid course. In the period from October, 1932 to December, 1933, however, there were 5 deaths among 23 consecutive cases. These 5 patients were adults and had much in common; with them circulatory collapse and anuria were the outstanding clinical features. They will be discussed in some detail later in the paper since it is the adult patient with neglected or unrecognized diabetes, brought for treatment in a state of shock with extreme dehydration and a low blood pressure, who presents the greatest challenge to the physician.

In the entire period since the last report[1], a total of 55 cases of diabetic coma have been treated (from October, 1932 to October, 1934). Twenty-seven of these were received in the first 9 months of 1934 and represent 2.8 per cent of the 961 diabetic admissions for all causes during this same period. On the average, between 2 and 3 patients with diabetic coma are received each month. These facts constantly impress upon us the need for redoubling efforts to eliminate this complication in the course of diabetes.

One cannot overemphasize the fact that diabetic coma is usually preventable and that its acquisition entails a physical insult to the patient as well as an economic loss to him and usually to the hospital. The average patient who acquires diabetic coma or the family of such a patient is not financially able to pay the hospital expenses let alone anything in the way of a doctor's fee. The total cost to a patient or to a hospital for the treatment of an attack of coma is not far from $100. The importance of this is made greater by the fact that certain patients are repeated offenders. In the present series of 55 cases, 4 patients (7686, 9233, 10550, and 12166) appear twice within the 23 month period studied. In the total series of 276 cases to date, 25 patients appear twice, 5 appear three times, and one patient four times, another five times, and still a third, six times. These 33 patients, then, have been responsible for 80 of the 276 instances of coma!

Palli and Waterhouse[2] have reported the case of a young girl (our one-time patient, 10505) who died in diabetic coma at the age of 16.8 years having had 22 hospital admissions in which acidosis, usually marked, was present. The duration of diabetes at the time of death was 8.6 years. Such chronic offenders present a great problem and demand the closest of supervision. Diabetic patients should be made to realize that modern treatment with adequate diets and insulin has placed in their own hands the responsibility for keeping well.

Responsibility of the Physician. Patients look to their family doctor for guidance in the proper care of their diabetic condition. This trust is a challenge to the knowledge and alertness of the physician. How tragic it is when he fails in his duty of diagnosing oncoming diabetic coma, or, recognizing it, fails to treat it early and adequately! The letter below describes just such a situation and needs very little comment. It is written by a wife regarding the death of her husband. This patient was first seen on January 6, 1922; onset of diabetes was in November, 1921 at the age of 27 years. The letter is published with her permission (the italics have been added).

October 16, 1934

My dear Dr. Joslin:

I have put off writing this note because it was hard to write, but I feel it is only fair to you and to other diabetics that I do it.

[Mr.] . . . died very suddenly last . . . He had been feeling especially well and had told me not ten days before his death that he had never felt better in his life.

I had had a slight attack of intestinal grippe and we thought that that was what [Mr.] . . . had when he went to bed nauseated on a Friday afternoon. He breathed very heavily and couldn't seem to get fresh air enough from then on, but none of us realized the seriousness of it. *Our doctor finally told me at midnight Saturday night that he thought it was sugar and that we might have to give him a quantity of insulin in the morning but he didn't even suggest giving it then.* About 7:30 Sunday morning there was a decided change in his looks and breathing and I called the Doctor quickly but [Mr.] . . . was gone in an hour and stimulants had no slight effect upon him.

I should like to know whether or not with the right treatment anything could have been done for him and also if the nervous condition might have brought up the blood sugar.

I have two children, 10 and 6 years, and if there is anything I can do to help them avoid diabetes besides being careful of sugar, I should be most grateful if you would suggest it.

Very sincerely,

*From the George F. Baker Clinic, Elliott P. Joslin, M.D., Medical Director, at the New England Deaconess Hospital, Boston.

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TABLE I

FIFTY-FIVE CONSECUTIVE CASES OF DIA

Case No.	Age at Coma, Years	Duration Diabetes, Years	Date	Clinical Data		Blood					
				Respiration	Mental Condition	Sugar, per Cent			Plasma CO_2 Combining Power, Volumes per Cent		
						Day			Day		
						1	2	3	1	2	3
			1932								
7711	11.3	3.8	Oct. 28	Kussmaul	Conscious	0.35	0.17	0.04	14	32	—
11502	32.4	10.4	Nov. 9	Kussmaul	Conscious	0.53	—	0.33	13	—	42
7686	17.8	3.8	Dec. 2	Kussmaul	Conscious	0.15	0.12	—	20	—	—
11531	38.5	1.4	Dec. 3	Kussmaul	Conscious	0.50	0.20	0.12	7	24	22
			1933								
9233	27.3	2.7	Jan. 13	Kussmaul	Conscious	0.63	0.27	—	7	32	—
8882	14.7	2.8	Jan. 23	Kussmaul	Conscious	0.30	—	0.12	9	—	48
11712	7.4	0	Feb. 18	Kussmaul	Conscious	0.41	0.16	—	20	—	—
6413	12.9	5.3	Feb. 23	Kussmaul	Conscious	0.65	0.26	—	15	36	—
7690	36.4	5.8	Mar. 20	Kussmaul	Unconscious	1.04	—	—	2	—	—
8059	15.9	3.8	Mar. 25	Kussmaul	Drowsy	0.44	0.04	0.04	13	—	—
3705	50.8	18.2	Apr. 24	Normal	Conscious	0.30	0.29	—	12	32	—
6287	17.8	11.0	May 4	Kussmaul	Drowsy	0.53	0.08	0.42	9	—	—
7735	26.1	4.5	May 21	Kussmaul	Drowsy	0.63	—	0.11	11	—	—
11920	51.2	0	May 25	Kussmaul	Drowsy	0.62	0.30	—	15	—	—
12014	43.2	2.3	July 21	——	Drowsy	0.30	0.17	—	17	—	—
12098	45.4	9.6	July 22	Kussmaul	Drowsy	1.15	—	0.05	10	—	—
6816	12.1	5.5	Aug. 18	Normal	Conscious	0.42	0.05	—	18	55	—
12139	56.9	0.1	Sept. 7	Kussmaul	Unconscious	1.07	—	—	19	—	—
12166	5.0	0.2	Sept. 19	Kussmaul	Drowsy	0.17	0.13	—	10	—	—
12208	12.9	0.1	Oct. 2	Kussmaul	Conscious	0.56	0.25	—	17	—	—
11033	30.7	2.3	Oct. 8	Kussmaul	Conscious	0.41	0.13	—	5	24	—
6920	49.5	8.9	Oct. 31	Normal	Conscious	0.38	0.17	—	11	—	—
12270	33.3	0.2	Nov. 5	Feeble: Gasping	Unconscious	0.64	—	—	18	—	—
9828	21.3	3.6	Dec. 3	Kussmaul	Conscious	0.49	0.23	—	12	—	—
10550	14.3	2.9	Dec. 19	Kussmaul	Drowsy	0.62	0.51	0.47	15	29	35
7210	71.8	5.7	Dec. 19	Kussmaul	Drowsy	0.61	0.14	0.13	8	33	49
6803	20.1	8.4	Dec. 19	Kussmaul	Unconscious	0.89	0.07	0.40	3	24	17
5029	36.3	8.7	Dec. 28	Kussmaul	Conscious	0.61	0.25	—	7	22	—
			1934								
12433	41.3	2.3	Jan. 16	Kussmaul	Conscious	0.26	0.15	—	16	39	—
12384	12.7	5.0	Jan. 17	Kussmaul	Conscious	0.26	0.16	0.13	13	13	—
12434	62.7	0.1	Jan. 17	Kussmaul	Conscious	0.53	0.05	—	13	40	—
10721	13.9	2.2	Jan. 21	Kussmaul	Conscious	0.42	0.29	0.04	16	37	—
11807	30.0	6.3	Jan. 25	Kussmaul	Conscious	0.22	—	0.29	18	—	—
12467	58.0	0	Jan. 29	Kussmaul	Drowsy	0.61	0.12	0.12	10	28	—
2786	53.3	11.6	Feb. 1	Kussmaul	Drowsy	0.53	0.05	0.06	10	31	36
12166	5.3	0.5	Feb. 1	Kussmaul	Conscious	0.49	0.04	—	11	20	—
11039	9.6	3.3	Feb. 17	Kussmaul	Drowsy	0.51	0.15	0.04	11	—	—
5319	13.7	8.3	Feb. 26	Kussmaul	Conscious	0.20	0.35	—	19	28	—
12608	62.1	0.7	Mar. 11	Normal	Conscious	0.50	0.22	0.12	20	—	—
8435	45.9	6.1	Mar. 12	Kussmaul	Conscious	0.54	0.35	0.02	13	—	—
7047	16.8	5.9	May 10	Kussmaul	Unconscious	1.05	0.13	0.42	4	10	13
12751	27.5	0.1	May 17	Kussmaul	Conscious	0.25	0.08	0.22	16	24	—
9376	28.6	4.5	May 18	Kussmaul	Conscious	0.49	0.09	0.29	6	31	—
9233	28.6	4.0	May 28	Kussmaul	Conscious	0.42	0.09	—	14	34	—
10550	14.8	3.4	June 4	Kussmaul	Drowsy	0.34	0.22	—	12	28	—
12887	48.8	0.1	July 2	Kussmaul	Drowsy	0.45	0.15	—	2	15	—
6533	33.6	6.7	July 5	Normal	Conscious	1.29	0.04	0.19	19	25	—
5926	11.2	7.4	July 7	Kussmaul	Unconscious	0.68	0.07	0.03	11	—	—
12922	5.5	0	July 26	Kussmaul	Drowsy	0.29	0.03	0.15	10	30	—
10719	61.8	2.8	Aug. 17	Kussmaul	Drowsy	0.44	0.11	—	8	41	—
6884	18.8	6.4	Aug. 25	Kussmaul	Conscious	0.80	0.12	0.06	9	37	—
12986	2.3	0.2	Aug. 27	Kussmaul	Conscious	0.41	0.33	—	11	—	—
9736	21.2	5.0	Sept. 9	Kussmaul	Drowsy	0.51	0.06	0.24	8	28	44
9029	7.8	4.4	Sept. 19	Kussmaul	Unconscious	0.24	—	—	13	—	—
7686	19.5	5.6	Sept. 20	Normal	Conscious	0.10	0.56	0.10	16	13	—

*These 20 patients had received before hospital a
an initial dose of insulin varying from 13 to 90 uni
insulin is not included in the total figure given in t
for the first day.

TABLE I

NSECUTIVE CASES OF DIABETIC COMA

Blood									Urine			Insulin Units			Comment and
Sugar, per Cent			Plasma CO_2 Combining Power, Volumes per Cent			Non-Protein Nitrogen, mg. per 100 cc.			At Entrance						
									Diacetic Acid	Sugar, per Cent	Sugar Free after Entrance, Hours				
Day			Day			Day						Day			
	2	3	1	2	3	1	2	3				1	2	3	
5	0.17	0.04	14	32	—	42	—	—	+++	3.6	7	110	40	41	
3	—	0.33	13	—	42	—	—	36	+++	5.2	9	245*	49	46	
5	0.12	—	20	—	—	33	33	—	+++	1.8	2	30*	40	64	
0	0.20	0.12	7	24	22	28	58	75	++++	3.4	15	403	145	—	Fatal. Bronc monia. A
3	0.27	—	7	32	—	—	26	—	+++	3.8	13	195	60	70	
0	—	0.12	9	—	48	30	—	—	++	3.6	18	105*	65	49	
1	0.16	—	20	—	—	—	—	—	+	8.0	4	34	17	21	
5	0.26	—	15	36	—	—	—	—	+++	5.6	9	80	39	47	
4	—	—	2	—	—	—	—	—	Trace	3.8	—	780*	—	—	Fatal. Anuri
4	0.04	0.04	13	—	—	—	—	—	++	5.6	8	150*	62	60	
0	0.29	—	12	32	—	25	—	—	++	3.0	10	125	25	35	
3	0.08	0.42	9	—	—	—	—	—	+++	5.4	15	120*	50	32	
3	—	0.11	11	—	—	32	—	—	—	—	17	246	28	65	
2	0.30	—	15	—	—	97	103	—	Trace	5.2	—	405	—	—	Fatal. Anuri
0	0.17	—	17	—	—	45	32	—	+	1.9	—	75	35	35	
5	—	0.05	10	—	—	91	—	61	++	3.4	20	505	55	60	
2	0.05	—	18	55	—	29	—	—	++	2.7	13	106	28	25	
7	—	—	19	—	—	118	—	—	0	5.2	—	450*	—	—	Fatal. Anuri
7	0.13	—	10	—	—	—	—	—	++++	3.5	7	97	20	22	
6	0.25	—	17	—	—	—	—	—	++	5.8	14	95	44	40	
1	0.13	—	5	24	—	34	40	—	+++	5.6	7	180	62	40	
8	0.17	—	11	—	—	31	—	—	++	3.7	—	135	59	62	
4	—	—	18	—	—	—	—	—	+	3.0	—	400*	—	—	Fatal.
9	0.23	—	12	—	—	—	—	—	+++	2.8	14	120	47	37	
2	0.51	0.47	15	29	35	—	—	48	+	3.0	11	145	46	41	
1	0.14	0.13	8	33	49	44	—	33	++	3.8	11	160	39	30	
9	0.07	0.40	3	24	17	47	—	29	+++	4.8	11	540*	100	55	
1	0.25	—	7	22	—	—	46	—	+++	3.3	16	190*	20	25	
6	0.15	—	16	39	—	31	—	—	++	2.5	4	70	35	35	
6	0.16	0.13	13	13	—	27	—	—	++	5.2	18	55	35	37	
3	0.05	—	13	40	—	56	37	—	++	3.9	10	140*	60	50	
2	0.29	0.04	16	37	—	—	38	—	+	2.4	11	99*	56	52	
2	—	0.29	18	—	—	47	—	36	++	2.7	3	95*	65	55	
1	0.12	0.12	10	28	—	63	64	34	Trace	2.8	9	295	105	37	
3	0.05	0.06	10	31	36	—	30	29	+++	3.0	—	195	80	60	Fatal. Pneu
9	0.04	—	11	20	—	—	—	—	++	4.7	6	120*	28	25	
1	0.15	0.04	11	—	—	—	—	—	++	6.0	11	175*	30	30	
0	0.35	—	19	28	—	—	36	—	0	0.5	10	45	55	53	
0	0.22	0.12	20	—	—	36	—	—	++	4.6	16	90	55	32	
4	0.35	0.02	13	—	—	—	—	29	+++	2.8	15	180	45	39	
5	0.13	0.42	4	10	13	—	49	—	+	3.8	15	615	80	50	Pulmonary T
5	0.08	0.22	16	24	—	30	25	—	Trace	0.4	14	130	50	55	Pregnancy.
9	0.09	0.29	6	31	—	—	47	—	+++	3.7	21	170*	40	50	
2	0.09	—	14	34	—	41	25	—	++++	4.2	5	104*	48	43	
4	0.22	—	12	28	—	—	35	—	+++	2.9	9	135	85	29	Hepatitis.
5	0.15	—	2	15	—	34	—	—	+	2.8	10	145	20	32	
9	0.04	0.19	19	25	—	136	102	97	0	2.5	—	515	15	120	Hepatitis.
8	0.07	0.03	11	—	—	—	—	—	+	2.6	10	260	70	30	
9	0.03	0.15	10	30	—	—	27	—	+++	5.6	19	110	45	12	
4	0.11	—	8	41	—	58	44	—	Trace	2.7	15	105*	46	36	
0	0.12	0.06	9	37	—	—	28	—	+	0.4	12	240*	90	72	
1	0.33	—	11	—	—	—	—	—	+++	6.0	—	23	14	12	
1	0.06	0.24	8	28	44	47	37	—	+++	1.7	8	340	76	65	
4	—	—	13	—	—	29	—	—	++	1.0	18	90	88	28	
0	0.56	0.10	16	13	—	—	39	—	+++	2.2	2	60*	57	62	

patients had received before hospital admission
e of insulin varying from 13 to 90 units. This
included in the total figure given in the table
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TABLE II

DIABETIC COMA

Summary Table—Comparative Study, by Averages, of the Six Series

Series and Number of Cases	Age at Coma, Years	Duration of D. M., Years	Date	Blood: Sugar, per Cent, Day 1	Blood: Sugar, per Cent, Day 2	Blood: Plasma CO_2 Combining Power, Volumes per Cent, Day 1	Blood: Plasma CO_2 Combining Power, Volumes per Cent, Day 2	Blood: Plasma CO_2 Combining Power, Volumes per Cent, Day 3	Blood: Non-Protein Nitrogen, mg. per 100 cc., Day 1	Urine at Entrance: Diacetic Acid	Urine at Entrance: Sugar per Cent	Insulin Units, Day 1	Insulin Units, Day 2	Insulin Units, Day 3	Fatal Cases: No.	Fatal Cases: Per Cent
I 24 Cases	31.5 (24)	2.4 (24)	May, 1923, to Mar., 1925	0.47 (23)	0.20 (20)	15 (24)	33 (20)	31 (10)	47 (16)	++++ (24)	3.3 (24)	154 (24)	63 (22)	58 (21)	5	21
II 28 Cases	31.6 (28)	2.8 (28)	Apr., 1925, to Feb., 1927	0.49 (28)	0.20 (20)	14 (28)	29 (20)	30 (10)	46 (21)	+++ (28)	3.4 (28)	166 (28)	59 (26)	49 (26)	4	14
III 53 Cases	29.1 (53)	4.0 (53)	Mar., 1927, to Feb., 1929	0.53 (52)	0.21 (46)	13 (53)	28 (37)	38 (12)	60 (23)	+++ (53)	3.1 (53)	183 (53)	40 (50)	39 (50)	5	9
IV 74 Cases	31.3 (74)	3.5 (74)	Feb., 1929, to Aug., 1931	0.49 (74)	0.20 (51)	11 (73)	29 (42)	35 (13)	48 (52)	++ (74)	3.7 (74)	252 (74)	49 (65)	41 (62)	13	18
V 42 Cases	26.1 (42)	3.5 (42)	Aug., 1931, to Oct., 1932	0.47 (42)	0.15 (37)	12 (20)	28 (28)	33 (5)	37 (17)	++ (42)	3.9 (40)	201 (42)	51 (41)	45 (39)	2	5
VI 55 Cases	28.5 (55)	4.3 (55)	Oct., 1932 to Oct., 1934	0.51 (55)	0.17 (46)	12 (55)	29 (31)	34 (9)	49 (28)	++ (54)	3.6 (54)	196 (55)	52 (51)	44 (50)	6	11

The subfigures indicate the number of determinations upon which the average is computed in each series.

This responsibility of the physician has recently been stressed by Dr. George H. Bigelow, Director of the Massachusetts General Hospital and formerly Health Commissioner of the Commonwealth of Massachusetts[5]. He states: "The unrecognized diabetic coma which still comes to the hospital is medically about as reprehensible as the case of diphtheria similarly unrecognized and untreated. The symptomatology which should arouse suspicion and the methods for precise diagnosis are to a certain extent analogous in the two diseases. The doctor unequivocably recognizes his responsibility in the diphtheria case and yet there is still too much complacency in regard to the neglected diabetic.

"It has been shown again and again that diphtheria mortality increases with the day of the disease in which antitoxin is first administered. Similarly in diabetes delay is the essence of failure. Prompt recognition and specific treatment must be recognized as vitally in diabetes as in diphtheria."

Clinical Material. In our last report[1] a summary was given of 221 attacks of coma in 189 patients* for the period from May, 1923 to October, 1932. To this number are now added the 55 cases (in 51 patients) in the present series, making a total of 276 cases in 228 patients* up to October, 1934. Eleven of the present group appeared in the earlier series.

In table I are recorded in detail the data regarding the individual cases, and in table II comparison is made between the present and the preceding groups of cases.

The average age of the patients does not vary much from year to year, being from 26.1 to 31.3 years; in the present series the average was 28.5 years and two extremes of age are seen: Case No. 12986 was only 28 months old, while Case No. 7210 was 71.8 years of age. Both patients recovered although the older patient has since developed pulmonary tuberculosis.

In the present series there were 18 males and 33 females. This preponderance of females becomes all the more striking when one considers that in the age group concerned, diabetes is more common in males than in females.

The duration of diabetes prior to the onset of coma was on the average 4.3 years which is the greatest of any series thus far. It is noteworthy, however, that in 12 of the 55 cases, coma occurred within less than two months after the probable onset of diabetes and in each instance the diabetic condition had been recognized only after the onset of symptoms of acidosis.

The average blood sugar on admission was 0.51 per cent; this had fallen on the second and third days to 0.17 per cent. A high blood sugar value does not necessarily imply a poor prognosis; in the present series there were 5 cases with an initial blood sugar value of over 1 per cent, and of these, three recovered and 2 died. To date among 276 cases of coma we have seen in all 10 cases in which the initial blood sugar value was 1 per cent (1000 mgm. per 100 cc.) or above; of these 6 recovered and 4 died. Haines and Davis[4] have reported 3 cases of diabetic coma (with recovery) in which the initial blood sugar values were above 1 per cent.

The average CO_2 combining power of the blood plasma was the same as that of the preceding series, namely 12 volumes per cent; this had risen on the second day to 29, and on the third to 34 volumes per cent. Five of the 55 cases had on admission a CO_2 combining power of 10 volumes per cent and 15 others a value below that figure.

The present series was notable in that 20 per cent of the cases had on admission a blood non-protein nitrogen of 45 milligrams per cent or over. The average value on the first day of treatment of the coma and the outcome of the cases are shown below:

Case No.	Blood Non-Protein Nitrogen, mgm./100 cc.	Result
6533	136	Recovered
10719	58	Recovered
11920	97	Died
12098	91	Recovered
12139	118	Died
12434	56	Recovered
12467	63	Recovered

Determinations of the blood non-protein nitrogen were not made in Case Nos. 7690 and 11270 but in these patients considerable nitrogen retention was in all probability present.

The average amount of insulin used in the first 24 hours was 196 units or practically the same as the corresponding figure for the preceding series. It is a noteworthy and encouraging fact that in 20 of the 53 cases of coma developing outside the hospital, an initial dose of insulin varying from 13 to 90 units had been given at home before leaving for the hospital. This had been given usually by the home physician, though occasionally by the patient or one of the family.

Mortality. The mortality of the present series was 10.9 per cent. This is considerably higher than the 5 per cent mortality of the preceding series but is less than that of all other groups previously reported except that of 1927-1929 when it was 9 per cent. The higher rate in the present series is due to the inclusion of 4 cases (described in detail below) which were admitted in a moribund or semi-moribund state after several hours of unconsciousness.

To the 29 fatal cases of coma listed in the report 2 years ago, must now be added the 6

*In the former series by mistake one patient who had more than one attack of coma was counted twice: 228 instead of 229, therefore, represents the correct total number of patients.

deaths in the present series. This brings the total to 35 fatal cases among 276 coma admissions in 228 patients, a case mortality of 12.7 per cent.

In the insulin-era one-ninth of all diabetic deaths in the hospital on the service of Dr. Elliott P. Joslin have been in diabetic coma. Since May, 1923 there have been 306 diabetic deaths; of these the 35 coma deaths represent 11.4 per cent. In table III the fatal cases are listed by years.

TABLE III

DEATHS IN COMA COMPARED WITH TOTAL DIABETIC DEATHS

New England Deaconess Hospital
May, 1923 to October, 1934
(Experience of Dr. Elliott P. Joslin and Associates)

Year	Total Diabetic Deaths	Deaths from Diabetic Coma	
		Number	Per Cent of Total
1923 (from May)	7	2	28.6
1924	19	3	15.8
1925	20	2	10.0
1926	18	1	5.5
1927	19	3	15.7
1928	24	3	12.5
1929	33	3	9.1
1930	39	4	10.3
1931	28	7	25.0
1932	41	2	4.9
1933	31	4	12.9
1934 (up to October)	27	1	3.7
Totals and Average	306	35	11.4

Quite recently Stafne[5] has reported the results of a study of deaths among diabetics in the state of Minnesota for 1931. He sent questionnaires to the physicians who in that year recorded diabetes as a chief or contributory factor of death on 547 death certificates. In 105 or 28 per cent of the 375 cases in which replies were received, diabetic coma was held responsible for death. Parenthetically it is of interest that "of the patients in these 105 cases, only sixty-six, or 63 per cent, received insulin at any time during the final illness when acidosis was developing and it was needed most urgently." Similar mortality statistics were found in the city of Duluth by Bartels and Blum[6]. Among 90 diabetics dying in hospitals in 1931, there were 24 cases, or 26 per cent, in which coma was the cause of death.

Influence of Age on Mortality. In the prognosis of uncomplicated diabetic coma two factors are outstanding: (1) the duration of pronounced acidosis before institution of treatment and (2) the age of the patient. Youth carries with it a great advantage. In the present series there were no deaths in patients under 30 years of age. Among the 276 instances of coma in the entire series, 73 were in patients under 15 years of age at the time of the attack of coma. Of these there has been only one death, and that was in a girl (9162), 14.1 years of age with diabetes of 3.8 years' duration, who entered the hospital in a moribund state after three days of nausea, vomiting, and abdominal pain at home. Even if one enlarges the group of coma cases among juvenile patients to include all instances occurring in patients under 20 years of age at the time of coma, one finds that there have been but 3 deaths among 128 instances of coma. This case mortality of 2.8 per cent contrasts sharply with that of 21.6 per cent among patients 20 years of age or over (32 deaths in 148 cases).

Table IV shows the mortality in the 276 cases by decades. The benefit conferred by youth is clearly demonstrated.

TABLE IV

INCREASING DANGER OF COMA WITH ADVANCING AGE

Age at Coma by Decades	Total Cases	Fatal Cases	
		Number	Per Cent
First	19	0	—
Second	109	3	2.8
Third	35	3	8.6
Fourth	31	5	16.1
Fifth	33	6	18.1
Sixth	31	13	42.0
Seventh	14	4	28.5
Eighth	4	1	25.0
Totals and Average	276	35	12.7

In a recent article John[7] stresses the necessity for early and adequate treatment of diabetic coma. His paper presents his experience with 218 patients largely in the first and second decades of life. Of these there were 31 instances of acidosis (in 12 of the 22 cases in which the plasma CO_2 was recorded, this value was *above* 20 volumes per cent) under his immediate care with 5 deaths. Of these 5 patients there was one each at age 11, 15.7, 16, 17, and 18 years of age.

Causes of Death During Coma and after Discharge from the Hospital. Including the 6 fatal cases now reported, there have been 35 deaths from coma in the entire series. Of the 193 cases who left the hospital after recovery from coma, 26 have since died (8 since the last report), 1 is untraced, and 166 are known to be alive. In table V below are listed the causes of death of the 61 fatal cases. Of the 26 cases dying after leaving the hospital following recovery from coma, 5 subsequently died elsewhere in another attack of diabetic coma and 4 died of tuberculosis.

The average duration of diabetes among the 35 fatal coma cases was 3.3 years and among the 26 cases dying after recovery from coma 7.3 years. The latter figure is high enough to show that despite one or more attacks of coma, the life expectancy of the diabetic is now steadily increasing. A still more encouraging fact is that the average duration of diabetes to January 1, 1934 of the 161 living coma cases who developed diabetes prior to that date is already 6.5 years.

TABLE V

CAUSES OF DEATH IN 61 CASES DURING OR FOLLOWING COMA (1923-1934)

Causes of Death	35 Fatal Coma Cases N. E. D. H.	26 Cases Dying After Discharge Following Recovery from Coma
Uncomplicated coma	11	5
Sepsis and metastatic infection	8	0
Gangrene	0	2
Cardiovascular disease	1	7
Cerebral hemorrhage	2	0
Pneumonia	7	1
Acute infections	0	3
Acute pancreatitis	3	0
Cancer	0	3
Syphilis	1	0
Tuberculosis	0	4
Hyperthyroidism	1	0
Toxemia from burns	1	0
Hypoglycemia	0	1

Of the 6 deaths reported now, 4 were in patients who were admitted to the hospital in extremis (7690, 11920, 12139, 12270). All 4 were unconscious or very nearly so, all had extremely low blood pressures, all were greatly dehydrated, and all secreted very little or no urine during the period of observation. Despite the use of very large doses of insulin and energetic measures to combat the condition of shock, little or no clinical improvement was seen and these patients died in from 4 to 13 hours after admission. The case histories follow in brief below:

1. Case No. 7690, female, 36 years of age with onset of diabetes in June, 1927, was admitted to the New England Deaconess Hospital on March 20, 1933 in an almost moribund state, having been totally unconscious for 12 or 13 hours. The blood sugar was 1.04 per cent and the CO_2 combining power, 2 volumes per cent. Despite 780 units of insulin given in the 7 hours after admission, the blood sugar fell only to 0.75 per cent and the CO_2 combining power rose only to 7 volumes per cent. The blood pressure which on admission was 42 mm. systolic (obtained by palpation) never rose above 70 mm. The output of urine became less and less. Salt solution was very poorly absorbed from the subcutaneous tissues. The respirations became more feeble and shallow and the patient died 7 hours after entrance. At autopsy no anatomical cause of death was found.

2. Case No. 11920, male, 51 years of age was admitted to the hospital on May 25, 1933. Sugar had been found in his urine 3 days before but no insulin had been given. On admission he was very drowsy, barely responding to stimuli. His pulse was scarcely perceptible and the blood pressure was only 70 mm. systolic. The blood sugar was 0.62 per cent, the CO_2 combining power 15 volumes per cent, and the non-protein nitrogen 97 mgm. per cent. With the use of 390 units of insulin over a period of 13 hours the blood sugar fell and the CO_2 combining power rose satisfactorily. Clinical improvement did not correspond, however, the blood pressure fell at times to 40 mm. systolic, he became anuric, and died 13½ hours after admission. Autopsy revealed a left hydronephrosis and hydroureter which may well have played a rôle in the poor response to treatment.

3. Case No. 12139, male, 56 years of age was admitted to the hospital on September 7, 1933 totally unconscious. He had begun to get drowsy at about 11 a. m., by 3 p. m. was in semi-coma, and was brought to the hospital at 8 p. m. Sugar had never been found in his urine until the admission specimen was examined at the hospital. He had been given 90 units of insulin just before leaving home. On admission the blood sugar was 1.07 per cent, the CO_2 combining power 19 volumes per cent, and the non-protein nitrogen 118 mgm. per cent. The urine (only 30 cc. obtained by catheter) contained 5.2 per cent sugar but no diacetic acid; this may have been an accompaniment of renal block. No urine was obtained through the catheter after the small initial collection. The blood pressure on admission was 54 mm. systolic and steadily fell despite treatment. The patient died 4 hours after entrance. At autopsy other than hypostatic congestion of the lungs, dilatation of the stomach, and slight degeneration of the adrenal medulla, no anatomical cause of death was found.

Richardson[8] describes a case of diabetic coma with a CO_2 of 7 volumes per cent who, nevertheless, had no diacetic acid or acetone in the urine. He discusses as possible causes for such a condition (a) the dehydration with accompanying decrease in the total base of the body, (b) the excretion of all the ketones as B-hydroxybutyric acid rather than as diacetic acid or acetone and (c) pathologic changes in the kidney which may impair the excretion of ketones. Labbé[9] likewise has called attention to the possibility of diabetic coma with a negative test for diacetic acid in the urine.

4. Case No. 12270, female, 33 years of age was admitted in a profoundly unconscious state on November 5, 1933. She had had "hard breathing" for 32 hours and had been unconscious for 16 hours. Diabetes had not been diagnosed until the day of admission. At first no blood pressure estimation was possible; later at a time of temporary improvement values of 58 to 66 mm. were recorded. The initial blood sugar was 0.64 per cent and the CO_2 combining power was 18 volumes per cent. With the use of 400 units of insulin there was chemical improvement and temporary clinical improvement. She rallied very little, however, then failed and died 5 hours

after admission. At autopsy there was bilateral congestion of the lungs and a septic infarct of the left lung. Death was thought to be due, nevertheless, to the diabetic coma.

These four deaths illustrate well the hazard of unrecognized and untreated diabetes, of prolonged unconsciousness before the institution of treatment, and the poor prognosis which must be attached to a low blood pressure, particularly if it fails to rise or falls despite supportive measures. It is possible that in certain cases of this sort blood transfusion might prove of value if given early enough.

Of the 2 other fatal cases of the present series, one was much like the 4 cases just described. This patient, Case No. 11531, male, 38 years of age, was brought to the hospital on December 3, 1932 with a blood sugar of 0.50 per cent, CO_2 combining power of 7 volumes per cent, and non-protein nitrogen of 28 mgm. per cent. With 403 units of insulin during the first 24 hours he improved both clinically and chemically. On the morning after admission he became anuric and the blood non-protein nitrogen began to rise. The blood pressure fell, twitching of the hands and facial muscles appeared, and the patient gradually failed and died in uremia about 50 hours after admission. The blood sugar was normal at the time of death. At autopsy, bronchopneumonia of the left lower lobe of the lung was found.

Case No. 2786, female, was 41 years of age in July, 1922 at the time of onset of diabetes. She was admitted to the hospital on January 31, 1934. Three days before, she had suddenly developed a sore throat. On the day of admission she had vomited repeatedly. The initial urine specimen showed 6.4 per cent sugar but the test for diacetic acid was negative. The insulin dosage given her in the next 24 hours proved to be inadequate in view of her infection. On the evening of the day following admission she showed unmistakable signs of diabetic coma and the diagnosis was confirmed by laboratory tests. With extra insulin and supportive measures there were satisfactory chemical improvement and excellent control of the diabetic condition but death took place 5 days after admission with pneumonia at both lung bases. No autopsy was obtained but the diagnosis was confirmed by roentgenogram.

It has been stated that of the 193 cases who left the hospital after recovery from coma, 26 have since died, including 8 cases since the last report. It is worth while to discuss further these 8 cases. Case No. 3078, female, aged 23 years, died at the New England Deaconess Hospital of pneumococcic meningitis following left otitis media. For months she had had pyelonephritis and had had several exacerbations of the kidney infection during which she was extremely ill. At the time of death her diabetes was of 11 years' duration (almost half of her life) and for the most part had been uncontrolled. Case No. 3750 died at the age of 39 of pulmonary tuberculosis with diabetes of 10.5 years' duration. A second death from pulmonary tuberculosis was that of Case No. 7486 who had diabetic coma when first seen in January, 1929; the diabetes was of 11 years' duration at the time of death. Case No. 4171, aged 75 years, died of heart disease 8 years after she had recovered from an attack of diabetic coma; the total duration of diabetes was 9 years. Case No. 6511 died of carcinoma of the breast at 55 years of age, 7 years after onset of diabetes. Case No. 8395, aged 51 years, broke diet, became lax in treatment and died at home in coma. Diabetes was of 7.3 years' duration. Since the previous report Case No. 6770, our oldest patient to recover from coma (72.6 years of age at the time) has died of coronary thrombosis. She was 77.3 years old at death. Case No. 7465 with diabetes of over 9 years' duration died of congestive heart failure at the age of about 56 years.

In studying 245 cases of pulmonary tuberculosis in diabetics, among the coma cases Root[10] found that 8 per cent developed tuberculosis within 3 years after recovery from coma. The effect of the metabolic disturbance in coma upon a chronic infection or the susceptibility to tuberculosis again emphasizes the tragic character of this complication.

Clinical Features. For a discussion of clinical features the reader is referred to previous reports[1]. Almost invariable accompaniments of full-blown diabetic coma are the nausea, vomiting and abdominal pain. McKittrick[11] in a recent article discusses from the surgical standpoint the differential diagnosis and treatment of a diabetic patient with such symptoms. It is well to recognize that a patient with diabetic coma may also have acute appendicitis and such rare cases should not be overlooked, but it is regrettable to have operated when the symptoms were due entirely to the acidosis.

Faulkner and Hamilton[12] studied in 15 cases the electrocardiographic changes which may take place in diabetic coma. Their surprising finding was that such changes were relatively slight and infrequent. On the other hand Klingenberg[13] reported that only 1 patient out of 10 admitted for diabetic coma had a normal electrocardiogram.

Etiology. That the chief causes for the precipitation of coma are preventable is well shown in table VI.

TABLE VI

ETIOLOGY OF DIABETIC COMA

1.	Inadequate treatment (Breaking diet, too little or no insulin, etc.)	23
2.	Infections	11
3.	Undiagnosed or recently discovered diabetes	12
4.	Cause not apparent (in most instances probably diet breaking or inadequate insulin dosage)	9
		55 Cases

Breaking diet or dropping of insulin or both still remain the commonest causes for coma. As far as the present series is concerned, table VII would seem to indicate that the patients followed treatment very well during the first year but after that showed increased laxity. Coma was most common in the period from 2 to 6 years after the onset of diabetes.

TABLE VII

DURATION OF DIABETES PRIOR TO COMA IN 55 CASES

Duration Diabetes Years	Number of Cases of Coma	Duration Diabetes Years	Number of Cases of Coma
0-1	14	6-7	4
1-2	1	7-8	1
2-3	8	8-9	4
3-4	6	9-10	1
4-5	4	10-15	3
5-6	8	15-20	1

This demands that education of the patient must not stop with the initial contact but that it must be a continuous process. The diabetic patient must be made to realize the value of careful treatment and encouraged to assume responsibility for himself.

There was a strikingly large number of patients with diabetes which had not been recognized or treated until the onset of symptoms of acidosis. The 12 such cases represent 22 per cent of the total. These cases represent a difficult group and it is only by education and stimulation of the interest of physicians and laity alike to the importance of early recognition of the disease that progress can be made. As is mentioned elsewhere, 3 of the fatal cases in the present series were from this group of 12 cases in which the diabetic condition was recognized only after the onset of coma. These three patients were then brought for treatment in a condition which proved to be hopeless. On the other hand 40 per cent had received treatment before admission and this is a far larger percentage than ever before and shows how knowledge of the disease and its treatment with insulin are spreading.

Of the 11 cases in which infections probably played a large part in causing the acidosis, there were 3 instances of upper respiratory tract infections, 2 of pneumonia, 2 of abscesses of the thigh, 2 of urinary tract infection, one of acute hepatitis and one of pulmonary tuberculosis.

In two cases (7735 and 11033) the patients had given up medical treatment and taken up Christian Science. Diabetic coma was the result. Fortunately both patients recovered and one (11033) has since been delivered by Cesarean section of a healthy, living child.

Complications. The present series is instructive particularly because of the complications. It has been stated that several patients had high values for blood non-protein nitrogen and 5 of the fatal cases were anuric in the few hours preceding death. Three patients had pneumonia (2786, 11531, and 6920); the first two died: case No. 2786 because of the quickly overpowering infection, and Case No. 11531 with uremia. Case No. 6533 had a prolonged course of acute hepatitis with coma developing in the hospital, and Case No. 10550 had toxic hepatitis apparently secondary to acute pharyngitis; both cases recovered. Case No. 12751 was pregnant when seen in coma in May, 1934 and in August was delivered of a healthy baby. When Case No. 7047, aged 16.8 years, was admitted in profound coma the temperature of 101.8° F. suggested some complication. Return to consciousness was slow and after recovery from the extreme acidosis the fever persisted. This was found to be contributed to by a left otitis media but due mainly to acute bilateral pulmonary tuberculosis. She was transferred to a sanitarium where now, five months after the attack of coma, she remains in a critical condition.

Treatment. Our present treatment may be summarized as follows:

(1) *Preparation Prior to Hospital Admission.* A patient in diabetic coma deserves to be in a hospital where every facility is afforded for constant observation, uninterrupted treatment including the giving of parenteral fluids, and as frequent analyses of the blood and urine as may be necessary. If one learns by telephone that a patient at home is to be brought to the hospital, a preliminary dose of 20 to 80 units of insulin given by the home physician may be advised provided the diagnosis seems certain. A bed at the hospital should be prepared and necessary equipment as blankets, hot water bottles, stomach and rectal tubes, salt and glucose solutions, insulin, and stimulants assembled so that when the patient arrives no time need be lost in instituting treatment.

(2) *Insulin.* On admission after the diagnosis has been verified by history, physical examination and examination of the urine, a preliminary dose of insulin of usually 20 to 100 units is given subcutaneously. This dose must be varied to suit the age of the patient, the degree of acidosis, and previous insulin administration. Occasionally, when acidosis is extreme or when there is circulatory collapse, an initial dose of 50 to 100 units of insulin may be given intravenously in addition to that given subcutaneously. Provided acidosis is marked enough and the blood sugar high enough, a dose of insulin similar in size to the initial dose may be given every half-hour until there is clinical and chemical evidence of improvement. Urine specimens are then secured at intervals of one or two hours with instructions to give insulin according to Benedict's test, for example, 20 units for a

red or orange test, 15 for a yellow, and 10 for a yellow-green test. This dosage, too, must be varied depending on the severity of the acidosis, the age of the patient, and the response to treatment. Unless absolutely forced to do so, we prefer not to catheterize patients for fear of resulting infection. Rather we make frequent blood sugar estimations (capillary blood may be conveniently used) as a guide to treatment if urine specimens cannot be secured.

(3) *Fluids.* One of the most striking clinical features of diabetic coma is the dehydration. The skin is dry and inelastic, the tongue and mucous membranes of the mouth are parched, the eyeballs are soft, and the subcutaneous tissues are obviously depleted of fluid. Atchley[14] and coworkers in a study of two diabetic patients with experimental acidosis have demonstrated this loss of intra- and extracellular body water and electrolytes which occurs following the withdrawal of insulin therapy and the reversal of the process when insulin is resumed.

Treatment must have, then, as one of the primary aims, the restoration of fluid and electrolytes to the body. This is best done by the subcutaneous administration (by gravity) of 1000 to 1500 cc. of normal salt solution within the first hour. If required, an infusion of 500 to 750 cc. of salt solution may be given intravenously later and indeed, a second or third subcutaneous infusion of 1000 to 1500 cc. may also be necessary within the first few hours. We have never observed embarrassment of the heart to follow even such liberal quantities of infusions. All solutions should be slightly above body temperature, and should be given slowly and with great caution to avoid infection or unnecessary trauma. Following a cleansing enema, salt solution may at times be given rectally with success. After gastric lavage, usually fluids as broths, orange juice, or ginger ale may be given cautiously by mouth. Usually it is preferable to wait perhaps an hour after gastric lavage and then begin by limiting such fluids to 100 cc. per hour.

(4) *Gastric lavage* should be carried out routinely unless the patient is in extremis or in such condition that the procedure involved would be dangerous. Usually one's efforts will be rewarded by finding the stomach filled with sizable quantities of fluid, food remains, and old blood. Removal of such contents and gentle lavage with warm water or normal salt solution relieves the abdominal distress, stops vomiting, and prepares the way for the early administration of fluids by mouth.

(5) *Circulatory stimulants* are practically never needed with children and in adults rarely produce startling or lasting results. Adrenalin in doses of 0.3 to 1.0 cc. may be given subcutaneously for extreme collapse. Ephedrine gives a more prolonged effect in raising blood pressure and may be given subcutaneously in doses of 0.5 to 1.0 cc. (25-50 mgm.). Either adrenalin or ephedrine may be given intravenously in emergencies but usually if the situation is grave enough to warrant such medication, the prognosis is bad.

(6) *Blood transfusion* was done in 2 of the cases (6803 and 7047) in the present series and both of these recovered. A false impression might be gained from this statement were it not realized that both these patients were young and that particularly in Case No. 6803, considerable improvement was noted just before the transfusion was begun. In former years occasional transfusions had been carried out in selected cases at the Deaconess Hospital and then, as now, it has always been extremely difficult to evaluate the benefit derived and to know whether or not the result obtained was influenced by the transfusion or by other factors. Sheppe[15] has recently reported good results from the use of transfusion in coma cases.

Peters, Kydd and Eisenman[16] state that the concentration of the blood seen in diabetic acidosis is due not only to the loss of fluid from the body but also to the loss of fluid from the blood vessels into the tissues. They believe that when, despite the usual treatment, improvement in the clinical condition does not take place, the blood pressure remains low, and the condition of shock continues, blood transfusion may be of great value in aiding restoration of blood volume. They employed blood transfusion in 9 cases of whom 4 died.

(7) *Food.* Usually glucose is not added to the subcutaneous or intravenous infusions used at the outset of treatment. However, such treatment may be of value if the blood sugar is relatively low and the acidosis relatively great. Very early we begin giving orange juice and ginger ale by mouth and try to provide an intake of at least 100 Gm. of carbohydrate during the first 24 hours of treatment.

(8) *A cleansing enema* is routinely given.

(9) *Alkalies* are not used. Experience has amply shown that alkalies are needless and if given in quantities necessary for effectiveness in a purely chemical sense, may be actually harmful. It is of interest that in a recent article Kydd[17] has lent support to the opinion that without alkalies, treatment with insulin, fluids, sodium chloride, and carbohydrate suffices to provide for recovery from diabetic acidosis and eventual restoration of body electrolytes.

Circulatory Collapse. Because of its importance a further word may well be said regarding the treatment of profound shock characterized by a blood pressure below 90 mm. of mercury (at times too low to measure), rapid pulse, and eventual anuria. This state is not limited to older patients and may be seen in advanced coma at any age. One must agree with Lande[18]

regarding the poor prognosis which attends such a case. On the other hand, we have seen unexpected recovery in a pulseless patient of 70 years treated with the methods outlined above. In this series Case Nos. 6803, 7047 and 10970 had blood pressures from 0 to 80 mm. but made good recoveries. Lawrence[19] has reported the use of gum acacia solutions and large amounts (4 to over 5 liters) of salt solution, both physiologic and hypertonic, intravenously to raise the blood pressure and counteract dehydration. Ralli and Waterhouse[20] stress the importance of large amounts of fluid. Labbé and Boulin[21] hold that the use intravenously of adrenalin 1 mgm. in 500 cc. of normal salt solution is of great value. Bertram[22] uses various circulatory stimulants including strophanthin, cardiazol, hexeton, coramin, strychnine, caffeine, and sympatol. In marked collapse he recommends the careful use of a constant intravenous solution of sympatol or adrenalin. We shall continue to use during the first 24 hours normal salt solution in amounts from 3 to 5 or more liters administered subcutaneously and intravenously in addition to fluids by mouth and by rectum. When indicated this will be supplemented by adrenalin or ephedrine subcutaneously or intravenously. The use of transfusion of whole blood will also be given further trial in cases with low blood pressure as under 70 mm. of mercury.

Hypertonic Salt Solution in Anuria. The present series of cases illustrates well that death from diabetic coma may be attended by anuria developing six to twelve hours before death. In the aged such anuria may be due to chronic nephritis but in the young it may be due to renal block caused by the acidosis. The repeated vomiting of coma may have so reduced the chloride of the plasma that urinary secretion ceases. Even when salt solution has been given subcutaneously, an analysis of the plasma chloride may show a low value. In 3 cases reported by Root[23] the injection of 50 cc. to 130 cc. of 10 per cent salt solution intravenously induced urination and relieved dangerous nitrogen retention. We did not fully appreciate this value of hypertonic salt solution in such cases until recently and did not use it in the coma cases reported in the present paper.

Summary. 1. Among 276 consecutive cases of diabetic coma in 228 patients, there were 35 deaths representing a case mortality of 12.7 per cent.

2. Of the 276 cases, 128 were in patients under 20 years of age and among these there were only 3 deaths, a case mortality of 2.8 per cent. In the entire series there has been only one death in a patient under 15 years of age and in the group of 55 cases now reported for the first time there were no deaths in patients under 30 years of age.

3. Among the 55 cases now reported there were 6 deaths, a case mortality of 10.9 per cent. Four of the 6 deaths were in patients (adults) who were admitted in profound shock after long periods of unconsciousness and who died within 4 to 13 hours after admission.

4. In 40 per cent of these 55 cases, treatment with an initial dose of insulin had been started, usually by the family physician, before the patient was brought to the hospital.

5. The 35 deaths from diabetic coma from May, 1923 to October, 1934 represent 11.4 per cent of the 306 total diabetic (hospital) deaths during this period.

6. In the entire series of 276 cases there were 10 cases with an initial blood sugar value of 1 per cent or above. Of these, 6 recovered and 4 died.

7. Circulatory collapse with a low blood pressure which fails to rise with the usual treatment indicates a poor prognosis. In such cases transfusions of whole blood may be considered in addition to the parenteral administration of large quantities of physiological salt solution and circulatory stimulants as adrenalin and ephedrine. In cases of anuria or oliguria with a low plasma chloride, the intravenous injection of hypertonic (10 per cent) salt solution may be of great value.

8. An attack of diabetic coma means a physical insult to the patient, particularly in that he is more prone to develop pulmonary tuberculosis subsequently, as well as an economic loss to him and the hospital. Chronic offenders present a great problem.

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IODO-BISMUTHATE OF QUININE IN THE TREATMENT OF SYPHILIS*

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THE use of bismuth in the treatment of syphilis was introduced by Sazerac and Levaditi[1] from the Pasteur Institute in 1921. Next to the discovery of the use of the arsenobenzenes by Ehrlich in 1909 this has been without doubt the most valuable contribution to syphilological therapeutics. Its superiority over mercury has been amply proved and its use where the arsenicals are contraindicated has prolonged many lives. Some workers have gone even so far as to put its value above that of the arsphenamines, such as Fournier at the Hôpital Cochin in Paris. In 1930 Levaditi[2] stated that after thirteen years studying the use of bismuth in syphilis, he found that its only point of inferiority to the arsenobenzenes lay in the more rapid disappearance of treponema of open specific lesions with the use of the latter, due to its quicker absorption through intravenous administration. "For all other purposes bismuth is superior to arsphenamine. Of equal curative value, sometimes even superior, bismuth has the inestimable advantage over arsphenamine of not exposing the patient to any very serious risks. Of course this holds good only if one follows . . . the exclusive use of insoluble or fat-soluble (soluble in oil) bismuth compounds and the use of suitable posology." American investigators have not gone so far as the French in their advocation of bismuth over arsphenamine, but its place in the treatment of syphilis is firmly implanted, as is evidenced by the management of the subject in all of the modern textbooks.

The number of bismuth products that are available have flooded the market in recent years, some forty odd having been brought forward. Probably the most complete classification will be found in the work of Schamberg and Wright[3]. Manifold papers acclaiming the attributes of various types of preparations have appeared. Most writers agree that the insoluble products, suspended in oil, give the best results with least pain and fewest complications. Among the more recent considerations of the subject are those of Beckman[4], Alderson and Ayres[5], and Harrington[6]. Which type or product will be found in a final analysis to be of the most value can only be determined by amassing all available statistics. To that end we are presenting some facts of interest.

The present study concerns the use of two of the insoluble bismuth salts in oil suspension, e.g., iodo-bismuthate of quinine and the more widely known bismuth salicylate. Fournier and Guenot (the first collaborators of Levaditi after his introduction of bismuth with Sazerac) brought out the iodoquinic salts of bismuth in 1921-1922, namely iodo-bismuthate of quinine (hereafter referred to as IBQ). This is the so-called "quinospirol" of Levaditi, who says, "From the first trials it was noted that this preparation, suspended in oil,—was endowed with remarkable curative properties and was perfectly tolerated by patients on intramuscular injection[2]." Since that time there has been very little written about the use of this particular compound. It has been used to considerable extent, however, in various parts of this country and abroad, especially in France[6]. Laubry and Bordet[7] advocated its use in the treatment of cardiovascular syphilis especially where they found as much and as rapid improvement as in cases treated with arsenobenzenes. Symptomatic relief was particularly noticeable. This preparation was less painful and did not cause stomatitis as readily as previous salts of bismuth in their hands.

Its use at the Massachusetts General Hospital was begun in 1925 and continued until 1932 when bismuth salicylate was substituted in the

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Out-Patient Department. IBQ has been retained, however, for use on ward cases throughout the hospital. We have not been convinced of the superiority of the salicylate either from the objective or subjective standpoint, in spite of the fact that it may be one of the most widely used bismuth products at the present time.

PROCEDURE

From a clinic with roughly seven hundred syphilitics under active treatment, a series of one hundred and ten uncomplicated cases were picked at random, without regard to the stage of the disease. No paretics, tabetics, cardiovascular cases or any with central nervous system involvement were included.

No obviously serofast cases were selected. All had a general checkup including serology and urine before a course of IBQ was begun. The course of heavy metals in this clinic constitutes fifteen injections. At the end a careful scrutiny was afforded each case. This included a complete physical examination with special attention to the integument, oral cavity and liver. The serology and urine were repeated, the latter also being done at least once during the course. The patients were questioned as to pain following injections, itching, skin eruptions, jaundice, stomatitis, urinary symptoms or gastrointestinal disturbances, any of which might not have been picked up in the routine interview each week. A goodly number had to be dropped from the list due to transfers to other clinics, intercurrent illness or skipping treatment; no case missing more than two weeks was included. The number finishing the course was thus reduced to seventy-six.

At the same time a similar series of cases was selected from the larger group of patients in the clinic as a whole, who were getting bismuth salicylate. The same restrictions were exactly adhered to throughout and the same type of case picked for each one receiving IBQ, i.e., according to stage, status of previous treatment and condition of patient, etc.

In each group there were six primary and thirteen secondary cases, which had all had previous treatment with the arsphenamines, as we did not feel justified in starting these cases on bismuth; they may better be designated as cases which had started treatment in the primary or secondary stages and are in the tabulation of results as "early" cases. Six cases diagnosed as tertiary had skin, mucous membrane or bone lesions as occurring in late infections. These with forty-one late and latent cases were grouped together under the heading of "late" as a matter of convenience, making a total of forty-seven. Separating these cases into each smaller division shed no more light on the situation. The remaining ten cases in each series were congenitals.

Serology tests done were the standard Wassermann and the Hinton test. In calculating the changes from this angle the following system was used: strongly positive Wassermann equals 4; moderately positive Wassermann equals 3; weakly positive Wassermann equals 2; doubtful Wassermann or a negative Wassermann with a positive Hinton equals 1; both Wassermann and Hinton negative equals 0; thus a gain or loss of four points was possible in each case. The Hinton test is read only as positive or negative. No case was found where the Wassermann was positive and Hinton negative, while many read Wassermann negative and Hinton positive; thus an additional hazard was provided before the case was called seronegative or "zero", in scoring. The complications occurring during treatment are recorded as such in the table.

RESULTS

The results of these two series are appended in table 1.

TABLE 1

RESULTS OBTAINED IN THE PRESENT SERIES

	Early		Late		Congenital		Total	
	IBQ	Bi-Sal.	IBQ	Bi-Sal.	IBQ	Bi-Sal.	IBQ	Bi-Sal.
Number of Cases	19	19	47	47	10	10	76	76
Serology*								
Gains	13	10	27	19	3	6	43	35
Unchanged	6	7	14	15	5	2	25	24
Losses	0	2	6	13	2	2	8	17
Albumin	0	2	1	2	1	0	2	4
Dermatitis	2	7	0	3	0	1	2	11
Bismuth Line	1	5	5	11	0	1	6	17
Stomatitis	0	2	0	1	0	0	0	3
Palpable Liver	0	3	4	4	6	2	10	9
Jaundice	0	2	0	1	0	0	0	3
Pain	5	12	9	27	0	5	14	44

*Figured on the basis of scores as explained above, the net points gained by the cases in the IBQ series was fifty-nine, whereas the net points gained in the bismuth salicylate series was thirty-one.

In addition to the present series of cases another group of patients was studied from the subjective angle. This consisted of one hundred and eighteen patients who had received both types of bismuth salt in the past and are still under observation. Their interrogation was facilitated by the variance in color of the products, one being a deep orange-red and the other white, making differentiation simple. In talking to these patients they were checked up by comments found on their records and nothing was included which was not of definite nature. The results obtained are listed in table 2.

TABLE 2

COMPARATIVE RESULTS OF 118 CASES FROM THE SUBJECTIVE ANGLE

	Iodo-Bismuthate Quinine	Bismuth Salicylate
Pain*	4	18
Dermatitis	1	4
Jaundice	—	3
Stomatitis	—	6
Gastro-Intestinal Symp.	—	2
Miscellaneous	—	4†
Completely Disabled	—	5‡

*Fifteen others complained of pain with both.

†One depressed mentally after each treatment.
One vertigo after each treatment.
One pains in joints after each treatment.
One numbness in hands and feet coming at end of course—cleared after the third injection of arsphenamine following.

‡Symptoms of this complication were severe enough to incapacitate these patients for two days or more; two of these had chills and fever.

COMMENT

The bismuth content of these two products is as follows: According to the originators, IBQ contained 23.85 per cent bismuth[2]. As computed by the Council on Pharmacy and Chemistry of the American Medical Association this product as marketed at present averages about twenty-five per cent of bismuth. Hanzlik and Mehrtens[8] found the average bismuth content of bismuth salicylate to be sixty-four per cent.

From these figures it would be expected that more pronounced results would be obtained with salicylate since it contained more than two and one half times as much bismuth. Our results, however, do not seem to bear this out. From the standpoint of the Wassermann there is a gain of approximately one hundred per cent more points with IBQ than with the salicylate, e.g., fifty-nine points versus thirty-one points. This cannot be explained satisfactorily from a strictly scientific viewpoint. The work of Hanzlik and Mehrtens[8] suggested that best results are obtained with bismuth in the anion form. Whether the IBQ assumes this form has not been worked out. These authors feel, however, that the insoluble compounds are objectionable. The presence of the iodine and quinine may play a part. The exact action of bismuth in combating this spirochete infection has not been established so that all such conclusions must be a matter of conjecture. Both products used here are in the insoluble group and are in oily suspension. The absorption and excretion of bismuth have been studied by a number of observers including Hanzlik and Mehrtens[8], Templeton[9], Thompson[10], Sollmann[11]. There is considerable variance in their reports. We have under consideration a repetition of these efforts in regard to the two products concerned here.

When the complications occurring during treatment are tabulated it is found that in regard to albumin the IBQ series exhibited two cases (2.6 per cent); the bismuth salicylate series four cases (5.3 per cent). No sediment examinations were done, which might have revealed still more difference. Eleven (14.5 per cent) of the salicylate cases experienced a dermatitis, while this was seen in only two (2.6 per cent) of the IBQ cases. The predominating types of eruptions seen were miliary follicular-papular and erythematosquamous eczematoid lesions. None were severe enough to interdict treatment. In the oral cavity seventeen cases (22.4 per cent) in the salicylate series evidenced a bismuth line at the gingival margins, three of which showed a definite stomatitis, whereas but six cases (7.9 per cent) in the IBQ series showed a bismuth line, with none having a stomatitis. It has been held by some that these gingival deposits are not a complication but merely a sign of saturation and are without harm. This is a moot point but certainly a stomatitis is not to be desired.

When the angle of hepatic damage is considered it is seen that IBQ produced one more case than bismuth salicylate, for the former series had ten (13.2 per cent) cases with a palpable liver and the latter but nine (11.8 per cent). However three of those receiving bismuth salicylate became jaundiced as well while none in the IBQ series did so. What part a preceding course of arsenicals may have played is controversial as is also the question of infectious jaundice. That these per cents of hepatic damage can probably not be ascribed entirely to the bismuth is suggested by the recent work of Irgang and Sala[12]. These writers did a very complete study from both the clinical and laboratory standpoints on two hundred and eighty-six cases under treatment with various types of bismuth. In no case did they find any clinical evidence of liver damage—not even a detectable jaundice. Ten per cent gave evidence of mild hepatitis as indicated by the positive direct delayed van den Bergh reaction. Liver function tests were not done routinely on all of our cases.

Perhaps the most important angle from the standpoint of the clinic patient is the pain and

stiffness persisting in the hip after treatment. In this respect it was found that forty-four cases (57.9 per cent) in the salicylate series were so affected whereas but fourteen (18.4 per cent) of the IBQ cases were so bothered—less than one-third as many. All injections were given by the same technician throughout. During the first half of the IBQ series the 0.2 gram dose was given in a dilution of 4 cc. of oil; in the second half it was given in only 2 cc. of oil —no subjective difference being complained of. It is worthy of additional mention that since these series have been finished we have had trouble with some of the IBQ cases in getting them to take a course of the salicylate later. Four of such have had severe enough local reactions to preclude the further use of bismuth salicylate; two were incapacitated for several days.

Still more interesting from the angle of the patients' personal comfort is the tabulation of results in the series of one hundred and eighteen cases which had in the past received a course of both of these types of bismuth. Here again the IBQ was found to have caused far fewer complications of any sort. Less than one-fourth as many had had pain with IBQ as with bismuth salicylate, e.g., four and eighteen cases respectively; an additional fifteen complained of this with both types of drug. With the exception of one case of jaundice all other complications were found only with the salicylate. These are clearly indicated in table 2 and need no further amplification. It is significant here again, however, that five of the cases having trouble during the treatments with bismuth salicylate were found to have been incapacitated for two days or longer.

SUMMARY AND CONCLUSIONS

1. Two parallel series, each consisting of seventy-six cases of uncomplicated syphilis were compared in regard to the results obtained with the use of two of the insoluble bismuth compounds.

2. The results as tabulated suggest that iodobismuthate of quinine, in spite of its lower bismuth content, is superior to bismuth salicylate.

3. An additional series of one hundred and eighteen cases which had in the past received both types of bismuth were studied from the angle of complications occurring during treatment. These cases were found to substantiate the results obtained in the present series, in that iodo-bismuthate of quinine produced a markedly smaller number of unfortunate incidents.

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WHAT WE HAVE LEARNED FROM ONE HUNDRED INTRAPLEURAL PNEUMOLYSES*

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THE most one may expect to accomplish by pneumolysis is to enhance the degree of lung collapse produced by artificial inflation of the pleural cavity when that collapse is insufficient to accomplish the intended effect because retarded by pleural adhesions; and occasionally to relieve pain or distress resulting from tension of adhesive bands.

In 1932 and 1933 there were seven hundred and fifty admissions to the Rutland State Sanatorium. In that period 314, or 41.8 per cent, were advised to have artificial pneumothorax. In 84 pneumothorax was not possible. Pneumothorax was performed on 230, or 26.5 per cent, patients admitted during that period. As approximately as could be determined, of 120 patients subjected to pneumothorax who were admitted during 1933, 80, or 66 per cent, have had pneumolysis performed. It is conservative to state that in over half of the cases of pneumothorax, pneumolysis is indicated.

Cutler[1] remarks "Of all forms of treatment in pulmonary tuberculosis, therapeutic or artificial pneumothorax occupies an increasingly

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important position and today it is practiced in approximately 50 per cent of all cases of tuberculosis."

Matson[2] states, "Pleuritic adhesions cause more failures in pneumothorax than any other complication, forty per cent of its failures being caused by such adhesions interfering with satisfactory collapse or compression of the lung." Further, "Intrapleural pneumolysis under thoracoscopic guidance will convert seventy per cent of the unsatisfactory type of pneumothorax into satisfactory ones."

Variation in type of pulmonary cases admitted in different institutions obviously accounts for a slight variance in the experience of different observers as to frequency of indication for collapse measures of any sort. However, it must be apparent to any student of pulmonary disease that adhesive pleurisy is an important factor to be reckoned with. It will be apparent then, that although the field of pneumolysis is restricted it is by no means a negligible one.

During the period from October 31, 1932 to January 15, 1934 inclusive, we performed 103 thoracoscopies with intent to sever pleural adhesions, at the Rutland State Sanatorium. In three it was deemed unsafe or otherwise inadvisable to cut adhesions; two of these patients were later subjected to thoracoplasty at the Massachusetts General Hospital with improvement; one who had had pneumolysis performed previously, at which time only a portion of the adhesions could be dealt with, was again subjected to a trial but no further cutting was considered safe. In the remainder, the adhesions were severed and they constitute our first one hundred intrapleural pneumolyses done at the above institution. While they were not strictly consecutive, we having operated similarly at other institutions, they were so far as the sanatorium is concerned, and are within our first one hundred and twenty-five operations of this type.

While not a large series of operations, one hundred is a sufficient number, when subjected to study, to indicate certain facts regarding the usefulness of the procedure, to emphasize the importance of certain sequelae, and from which useful conclusions may be drawn.

Sex.—Fifty-six of the one hundred operations were upon female patients, and forty-four upon males. The sexes of the patients in the institution are about evenly divided.

Age.—The youngest patient was seventeen; the oldest forty-three. Thirteen were aged from seventeen to twenty; sixty-six from twenty-one to thirty; twenty from thirty-one to forty; and but one over forty.

Stage of Disease.—Six were recorded as "Stage I," fifty as "Stage II," and forty-four as "Stage III."

The general condition of the patient was recorded as "good" in forty-three, "fair" in forty-seven, and "poor" in ten.

The sputum before operation was positive to tubercle bacilli in seventy-seven; negative in twenty; and not stated in three. After operation it was positive in twenty-two; negative in seventy-seven; and not stated in one. The amount of sputum was decreased after operation in sixty-four; increased in five; not changed in twenty-five; and the record not clear in six. Before the operation the sputum was scanty or absent in twenty-six; after operation in fifty-six.

The apparent onset of symptoms of the disease as noted in the records was within the period between 1920 and 1929 inclusive in twenty-two; from 1930 to 1933 inclusive in seventy-eight.

Interval.—The period between beginning therapeutic pneumothorax and the performance of pneumolysis averaged one hundred and ninety-eight days (slightly over six months). Inasmuch as there are included in the series several cases that had been resident in the sanatorium and under pneumothorax treatment for considerable periods when the earlier pneumolyses were done, this figure cannot be accepted as the average for the present time, which is probably considerably less than six months. As to how long one should wait after pneumothorax is instituted before performing pneumolysis, cannot be arbitrarily stated. The probability of a satisfactory therapeutic result with the collapse already obtained, the character and number of adhesions, and whether they may be expected to relax to a degree permitting collapse of actively diseased tissue and closure of cavities that may be present, extent of disease, reaction to pneumothorax, general condition of the patient and many other factors are to be considered. However, it is our inclination to doubt the wisdom of long-continued efforts to stretch adhesions, running into periods of many months, especially with positive manometric readings. It is our opinion that, under these circumstances, the danger of inducing bronchopleural fistula with its serious results is not inconsiderable. On the other hand, the mere presence of adhesions is not an invariable indication for action and time should of course elapse sufficient to observe and evaluate any reaction to inflation and note any improvement that may result from the partial collapse.

All adhesions present were severed in forty-seven pneumolyses. This was impractical in fifty-three. The principal reason for not completing pneumolysis at one sitting was the presence of areas of close adherence between the visceral and parietal pleurae. Other reasons were vascularity or bleeding, poor visibility, fatigue of patient, and inadvisability of unduly prolonging operation.

Complete severance of all adhesions may not be necessary for satisfactory collapse, but often we may be obliged to content ourselves with improved collapse. Frequently short bands, or close adhesion, left after partial pneumolysis may become stretched sufficiently to be successfully dealt with at a later operation, after improved pneumothorax. A perfectly efficient collapse may result even though a few adhesions remain.

The immediate convalescence was absolutely uneventful in twenty-eight cases. Postoperative fever of 101 degrees Fahrenheit, or over, occurred in twelve. This usually was transient, resuming normal in from one day to one week. Dyspnea was noted in five cases, one of which was accompanied by cyanosis. Tachycardia occurred twice. Cough was markedly increased for a time in one case. Pain in the chest, shoulder, arm, or back occurred postoperatively in nine. One patient developed a transient generalized jaundice. There was one case of hydropneumothorax. Faintness and dizziness were complained of in three instances and nausea in eight, resulting in vomiting in three.

Of immediate untoward sequelae of operation, the most important is bleeding. This usually results from severed vessels in the adhesion itself, the blood escaping usually from the parietal segment or from injury to intercostal vessels. It is possible to injure an intercostal vessel while introducing the trocar resulting in escape of blood into the pleural cavity.

We encountered considerable bleeding in one case; moderate bleeding in two cases; slight negligible bleeding in two or three cases. We met with no hemorrhage that could not be controlled by application of dull cautery or electrocoagulation. However, one can readily conceive of serious hemorrhage unexpectedly occurring and meticulous care should be taken to avoid it. Any adhesion appearing vascular should either be coagulated before cutting or avoided altogether, and facilities should be at hand at all times for dealing with hemorrhage. In its presence, one should work rapidly, but positively, and with presence of mind in order that it may be under control before the field of vision may become obscured by blood. Judgment and apprehension should attend the severance of thick or close adhesions. Hemorrhage from vessels not accessible to ligation may cause any surgeon concern wherever encountered.

Emphysema of the tissues about the wound occurred in forty-two instances. It was slight in fifteen, moderate in sixteen and considerable in eleven instances. Among the latter were some where crepitation was noted on both sides of the chest and neck and in one it was general over the trunk, extending down to the groins. Several times escape of air threatened loss of collapse. This actually occurred, completely, in four instances, partially in four others and once it became complete six months after operation. We believe this misfortune should happen less frequently in the future. Experience has taught us a better closure of the trocar wounds and the desirability of early fluoroscopy and when indicated, prompt and if necessary, frequent refills. Emphysema was invariably evanescent and inconsequential except in so far as accompanied by threatened or actual loss of collapse.

Fluid appeared in the pleural cavity, as shown by x-ray, either quite promptly or some time after operation in fifty-two instances. The amount was recorded as "slight" in sixteen, "moderate" in thirty-one, and "considerable" in five. It was known to be bloody in a few cases and it was recorded as present before operation in three.

Empyema.—Included in the above were seven pleural effusions which became purulent. Usually empyema was a later development of, or following, surgical procedure, but in a few it occurred rather promptly. It is an important and unpleasant sequel, although not invariably incompatible with ultimate recovery. It has usually occurred, in our experience, in otherwise bad cases. Some factors in its production probably are not within the surgeon's control. Selection of case, avoidance of bleeding, which might result in hemothorax, avoidance of release of infection by wounding the lung, and meticulous asepsis in technique of both operation and subsequent pneumothoraces, may be considered prophylactic measures to be observed. Of the seven, the sputum became negative to tubercle bacilli, or absent altogether, in five, and continued positive in two. Oleothorax, using gomenol oil, was done in three, with apparent noticeable improvement in two. The benefit in one was doubtful. In two empyemas, pleurocutaneous fistulae resulted. One patient, however, has at the time this is written, no cough or expectoration and has gained twenty pounds in weight, notwithstanding the fistula still present. Three of the seven have done poorly and the prognosis is bad. Two are doing well clinically and the prognosis is good. In two, while there is some evidence of improvement, the prognosis is problematical. Guinea-pig inoculation showed the presence of tubercle bacilli in the pus of all seven cases. Cultures indicated the presence of other pathogenic organisms in two and in one, with persisting pleurocutaneous fistula, mixed staphylococcus infection developed eventually.

Bilateral Cases.—There were fourteen intrapleural pneumolyses done upon nine patients who were subjects of bilateral pneumothorax. In one patient, we severed adhesions twice in the left pleural cavity after a pneumolysis had been done, by another surgeon, on the right. The general condition of the patient prior to operation was recorded as good in only two,

fair in six and poor in one, the operation proving a life-saving measure in the latter. In this group are included some desperate cases whose future prospects were nil without successful collapse. The more radical measures were contraindicated and we felt that pneumolysis should be done regardless of the obvious fact that the general mortality rate of the operation might be unfavorably affected. Marked clinical improvement in four of the nine patients, with some at least temporary if not permanent improvement in four others, surely justifies the one fatality. We are informed that one of these patients, a lad of seventeen, has since succeeded in joining the United States Marines, of course unsanctioned by his medical advisers at the sanatorium.

Clinical Results.—While from time to time we have encountered certain annoying sequelae of intrapleural pneumolysis, the importance of which we do not wish to minimize, we are able to report eighty-five per cent of the series as doing well at the time this is written. Marked improvement, obviously resulting from the procedure, occurred in fifty-three. In thirty-two others the patients are doing well, either because of less striking benefit of operation, or regardless of it. In fifteen it was evident that no benefit resulted, and, of them, seven were apparently made worse by the operation. In the latter number are included two followed by death within the period of six weeks. In neither was the prognosis at all good. One we consider an operative fatality. In evaluating the clinical results of pneumothorax, supplemented by pneumolysis, one should not lose sight of the bearing of disease in the contralateral lung, on persistence of tubercle bacilli in the sputum, or on the patient's prospects. Of course, in some of the cases showing less striking improvement, this might have occurred had not operation been done, other than the imperfect pneumothorax. We sometimes wonder if we do not easily forget the good recoveries that not infrequently resulted from sanatorium régime when the treatment of pulmonary tuberculosis rested so gracefully upon that tripod whose three legs were rest, fresh air, and nourishing diet.

As to technique, our present preference is for an electrocautery one, not however exclusively, for we consider it wise to coagulate before cutting certain obviously vascular adhesions and this is best done by means of the high frequency electrodes. We prefer for most cases the Jacobaeus-Unverricht instrument. Occasionally we have used the Cutler-Davidson jaw-electrode thoracoscope and while in some cases it is admirable, we do not find it always, in our hands, well adaptable. Rarely do we find cases in which there are multiple adhesions where all can be severed, even if seen through a single cannula, without change of position of puncture. We have resorted to the use of a second cannula. With this instrument, one is of course confined to the use of the high frequency current. The calibre of the instrument is slightly larger than the Unverricht and therefore because of larger incisions and punctures, more favorable to escape of the pleural air. Because of the facility with which bleeding points can be controlled and sometimes prevented by high frequency coagulation, we would not think of operating without an assortment of the electrodes at hand together with a suitable unit. There are several efficient electrosurgical outfits now upon the market, both tube and spark gap, or combined, at reasonable cost, which are perfectly suitable for this work. The electrocautery and the high frequency knife both have their devotees. Those who become familiar with one or the other probably do best work with their choice and as Sir Berkeley Moynihan says, "Surgeons, also, wear their rue with a difference."

There are, however, two phases in the use of high frequency cutting instruments in this work that ought to be at least considered by those unaccustomed to them. First, while the cutting is done with great facility and this is pleasing and attractive, a sudden motion on the part of patient or operator, not anticipated, may bring the point of an active electrode in contact with tissues to which one may wish to avoid injury and unintentional solution of continuity result. Accidental injury is of course less likely with long experience with pleural endoscopy, but experience involves many patients. Secondly, it is our conviction that sloughs resulting from deep injury to tissues are more readily produced by high frequency than cautery electrodes.

John R. Caulk[3], attempting to prove the supremacy of the electrocautery to the high frequency loop in transurethral prostatic resection, has in conjunction with Mr. Wilbur Harris, in the Department of Surgery at Washington University, conducted a series of experiments using a standard thermocouple galvanometer. The thermocouple placed in the tissues at varying distances from the active electrodes, appeared to show that a much greater intensity of heat occurs within tissues in the path between the high frequency electrodes than occurs at similar distances in any direction from the cautery. One of Caulk's deductions was that "high frequency currents produce heat in tissues away from the site of actual burning, often beyond the thermal death-point of the tissue; that cautery heat does not penetrate to such depths, its only heat resulting from conduction and is superficial." The principles governing high frequency cutting and coagulation are the same when applied to intrapleural or any other tissues as when applied to the prostate. It is not our purpose to condemn the use of the high frequency current for this work, but to emphasize its potential dangers that they may be kept in mind by those beginning its use. For

several years we have used the electrosurgical knife and surgical diathermy, where we considered it indicated, with satisfaction, and consider it admirable when not misapplied.

Some thoracic surgeons have found an accessory illuminating device whereby a small electric bulb may be carried through one of the Unverricht cannulae beneath and about adhesions during exploration, while at the same time looking through the thoracoscope, passed through another. The McCarthy foroblique telescope, which we have at hand as a part of the Cutler-Davidson instrument, serves this excellent function, with the additional advantage of a forward accessory view when desired. Being a delicate instrument, care is necessary to avoid breaking off the bulb or bending the shaft. Of course, it can not be used in conjunction with a curved cannula.

Case Reports

A. S., aged twenty-four, male, salesman, Stage III. Disease—upper two-thirds right; upper third left. General condition fair; no complications.

Onset catarrhal in March, 1930. Artificial pneumothorax started soon after admission January 22, 1932. April 26, 1932 pneumothorax started on opposite side. Failed to show improvement; collapse good on left, but unsatisfactory on right. Large cavity kept open by dense adhesions.

Prognosis: Guarded.

January 16, 1933. Pneumolysis right, not completed.

February 3, 1933. Pneumolysis right, completed.

After second operation, patient became dyspneic. Generalized emphysema. Dyspnea increased, fluid appeared. The second day, i.e., February 4, 1933, 1600 cc. air aspirated. February 6, 1933 pressure positive. A large needle was left in second interspace anteriorly. There was perforation of the lung. The patient continued to get worse and died February 13, 1933.

The operation seemed to hasten death and the fatality should be charged to it, although the future of the patient was unpromising in any event.

No permission was secured for autopsy.

E. K., aged twenty-one, female, shoe worker, Stage III.

Onset of illness insidious December, 1929. Physical signs indicated disease in entire left and upper half of right lung. General condition poor. Complication, kidney disease. Raising one ounce positive sputum.

Prognosis: Very poor.

Artificial pneumothorax was done on left side as a last resort measure. An unsatisfactory degree of collapse resulted and the patient was referred to me for thoracoscopy.

Operation September 25, 1933, left. A line of close adhesions to parietal pleura was found running around the apex of the lung. Web-like portions were released, but it was not possible to release the apex from chest wall. A thin-walled cavity was obviously in immediate contact. Operating time: thirty-six minutes.

After the operation, the patient complained of nausea and pain. Considerable subcutaneous emphysema. Escape of air necessitated the introduction of 300 cc. of air to prevent complete expansion. A few days following, fluid appeared continuing to increase in amount. In October, severe attacks of dyspnea occurred accompanied by cyanosis and a feeling of tightness over the epigastrium. Pulse was 140 and upwards during these attacks and the fever became 102° and 103° Fahrenheit. She developed definite signs of peritonitis. October 28, 1933, 500 cc. of air was withdrawn. She died on October 30, 1933.

No autopsy was performed.

We do not consider this an operative fatality, but the case is detailed inasmuch as the death occurred within the subsequent six weeks.

One might question the wisdom of operating on cases of this gravity. In such conditions as diffuse peritonitis from ruptured septic appendix or of hollow viscera, although the patient's prospects are almost nil, no one doubts the justice of a trial of surgical intervention. We ought to apply the same reasoning to surgical methods of collapse of the lung in advanced phthisis, if there appears to be a remote chance. Fortune will deal kindly with us once in a while in either case, but in neither ought surgery to be withheld until advanced disease supervenes. However, sometimes we do not have the privilege of early contacts.

We conclude that intrapleural pneumolysis is a valuable adjunct to therapeutic pneumothorax. It is a delicate procedure, requiring patience and gentleness in its performance. While continued experience enables one to avoid some of the unpleasant and untoward sequelae, occasional, if uncommon, disasters, notably empyema, will have to be reckoned with when using any of the present techniques.

I wish to express my thanks to Dr. Gabriel Nadeau of the Sanatorium Staff for searching and abstracting the records.

TUBERCLE BACILLI IN SPUTUM

	T. B. Pos.	T. B. Neg.	T. B. ?	Total
Before Pneumolysis	77	20	3	100
After Pneumolysis	22	77	1	100

CLINICAL RESULTS

Marked improvement	53
Others doing well	32
Not improved	15
Total	100
Made worse	7
Died as result of operation	1
Died within six weeks, not of operation	1

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CASE RECORDS
of the
MASSACHUSETTS GENERAL HOSPITAL

ANTE MORTEM AND POST MORTEM RECORDS AS USED IN WEEKLY CLINICAL-PATHOLOGIC EXERCISES

EDITED BY RICHARD C. CABOT, M.D.

CASE 21071

PRESENTATION OF CASE

A fifty-seven year old American housewife entered complaining of diarrhea.

Approximately nine months before entry she was seen at her home because of mucus and blood in her stools and diarrhea of about four months' duration. A rectal examination was not entirely satisfactory at that time but it was felt to be negative. She was not seen again until four months later at which time she reported one or two normal movements a day with a slight amount of mucus and occasional blood. A barium enema at that time showed an incompetent ileocecal valve and stasis in the colon, which was smooth. She was put on bismuth and tincture of belladonna with no success. Examination of the blood two months later showed a red cell count of 4,000,000, with a hemoglobin of 65 per cent. The white cell count was 6,300. She was given a low roughage diet and iron. A proctoscopic examination at this time was said to be negative. Two months before entry another proctoscopy showed an inflamed mucosa throughout the rectum with small red bleeding points. There was no evidence of malignancy. She went to bed the following day and was given iron and liver extract. The temperature was about 100° to 101°.

One week later she complained of nausea and vomiting. At that time she was admitted to another hospital. Her stool examinations showed some mucus but no blood. No amebae or pathogenic organisms could be found. Her red cell count remained around 3,500,000, with a hemoglobin of 60 per cent. The white cell count ranged between 7,000 and 11,000. The temperature remained slightly elevated. Her pulse remained around 120. The abdomen was somewhat spastic on the right side. She was given a high vitamin diet. She was discharged from the hospital after about two weeks. At home she felt somewhat better although she continued to have three or four stools a day. She continued to have a moderate degree of anemia, and about ten days before entry she was given liver extract for a period of seven days. During this time the stools were reduced somewhat in number and were definitely formed rather than loose. She was also started on iron. The evening before admission she developed nausea and the diarrhea became very severe.

A complete physical examination was not done at this time. Her abdomen was rather full, rounded and non-tender, the right side of the abdomen being rather more prominent than the left. Her general condition was poor.

The temperature was 101°, the pulse 120. The respirations were 20.

Examination of the blood showed a red cell count of 3,450,000, with a hemoglobin of 55 per cent. The white cell count was 5,500, 79 per cent polymorphonuclears. The stools continued to show blood and mucus but no amebae. A gastric analysis showed no free hydrochloric acid but a total acid of 5.

On the third day she was given a transfusion of 500 cubic centimeters. A barium enema showed no evidence of obstruction at any point. The walls of the colon were rigid and there was complete absence of haustral formation and of normal mucosal markings. The margins of the large bowel showed a saw-tooth appearance.

She continued to receive Blaud's pills, liver extract and Harris yeast. On the twenty-fifth day an ileostomy was performed. She did very well for three or four days but then began to have nausea, vomiting, some loss of appetite and elevation of pulse. She was transfused again and seemed to be getting along fairly well. About two weeks after operation she began to do poorly again. Two days later she developed abdominal discomfort and passed large quantities of blood through the ileostomy. No bleeding point could be observed, and it was assumed that it came from the large bowel, back through the short segment of ileum, between the ileostomy and the cecum. She failed rapidly that day and died early in the evening.

DIFFERENTIAL DIAGNOSIS

DR. ARLIE V. BOCK: In contrast to Dr. Miller's case*, I think you will agree with me, as this case is read, that the picture as given is indefinite and unsatisfactory. I get the impression that the people in charge of her never did get down to brass tacks in the study of the patient until at least the last event.

The first few lines of the present illness bring up the differential which has been discussed by Dr. Miller of ulcerative colitis, amebic and bacillary dysentery. It does not say whether the patient has been out of New England but, whether or no, the diagnosis of ulcerative colitis, with this story of diarrhea, mucus and blood, is the most likely.

Sometimes it is difficult to do a rectal examination, especially if the anal canal is involved as it may be in ulcerative colitis.

"She was not seen again until four months

*To be published.

later at which time she reported one or two normal movements a day with a slight amount of mucus and occasional blood." That suggests a remission of the underlying condition of ulcerative colitis which we know occurs in a great many cases of relatively low-grade type.

The appearance of the colon by x-ray is highly suggestive of the above mentioned diagnosis.

"She was put on bismuth and tincture of belladonna with no success." It is hard to judge what they mean by "success", because at that time she was having very little diarrhea.

With a diarrhea of only one or two bowel movements a day she appears to have been in an inactive state and proctoscopy might not have shown very much, although one might have expected evidence of healed ulcers. Active ulcerative colitis may occur without diarrhea. The history is lacking in that during the interval between proctoscopies there may have been an exacerbation of symptomatology.

The blood picture is one that is quite commonly found in any of the dysenteries, including ulcerative colitis. There is usually a hypochromic anemia but hyperchromia may occasionally occur.

"The abdomen was somewhat spastic on the right side." That is a finding not uncommonly found in cases of ulcerative colitis, presumably due to the tendency of the lesions to invade the wall of the colon.

I should think from the story that she was not kept in the hospital long enough; but that may have been a matter over which the doctors in charge had no control.

A complete physical examination is not recorded. We have no picture of what she looks like; no history of weight loss; no statement as to the appearance of her tongue.

The blood showed the same picture as previously.

If this were a case of amebic dysentery I should think they would have had little trouble in finding amebae. If one is uncertain of the diagnosis it may be useful to try the effect of a course of treatment with emetin.

In a great many cases of diarrhea, from any sort, we have either absent acid or very low acid in the stomach. It is not specific for any one of these conditions.

Much is left to the imagination in the description of this case but there is evidence of fairly long standing chronic disease which is characteristic of ulcerative colitis. It does not suggest sprue or any deficiency state.

I presume she died largely as a result of the effects of hemorrhage and presumably from ulcerative lesions of the colon. I find it very difficult with this type of picture to make a guess at anything besides ulcerative colitis.

X-ray Interpretation

Dr. George W. Holmes: In these cases I think it is probably worthwhile to take a film of the chest because a certain number of the tuberculous types do show evidence of a lesion in the lungs. A positive finding in the lung would be of some help, a negative finding of no value.

These films show a rather large, active small bowel, and in the large bowel there is absence of haustral formation; no normal mucosa; a narrow spastic bowel. Those two films show the condition fairly well. One, with the colon filled, and one, after evacuation.

Clinical Discussion

A Physician: The summary of the clinical record does not give a clear picture of this case especially as regards treatment. There was a long discussion as to whether we were dealing with true ulcerative colitis, which should be treated by ileostomy, or whether the condition was primarily a deficiency disease which could be cured by liver extract and high vitamin feeding.

Dr. Bock: I do not quite see the argument about the case. We know from observation of chronic ulcerative colitis that many of them show evidence of deficiency states. Dr. C. M. Jones presented several cases three years ago. There is a discussion in one of the recent *American Medical Association Journals* about deficiency and ulcerative colitis. Occasionally there are eye changes; the tongue is a famous example of deficiency disease. Anemia of the hypochromic type is common. Occasionally there may be enough disturbance to produce nutritional edema. I should think that the proper method of therapy is to give a high-vitamin low-roughage diet, including the use of liver and iron, and to consider ileostomy early rather than late in the disease.

A Physician: One of the consultants said that he had seen colons just as bad as this cured on liver treatment, and I believe that in his opinion this was a primary deficiency disease with secondary changes in the colon.

Dr. Thomas V. Urmy: This was not an acutely ill case until about the time operation was done. It was a low grade, chronic affair about which at first, at least, there was some doubt as to whether a true colitis was present. Though it may seem to illustrate the usual bad results of too long postponement of ileostomy, I am not sure that in this particular case the termination would have been influenced by earlier operation.

Clinical Diagnoses

Ulcerative colitis.
Intestinal hemorrhage, ? perforation.
Ileostomy.

Dr. Arlie V. Bock's Diagnoses

Ulcerative colitis.
Intestinal hemorrhage.

Anatomic Diagnoses

Colitis, chronic ulcerative.
Mesenteric thrombosis, venous.
Operative wound, ileostomy.
Fatty vacuolization of liver.
Pancreatitis, chronic.
Pulmonary emphysema, slight.
Pulmonary tuberculosis, healed, apical.
Pleuritis, chronic fibrous.
Arteriosclerosis: slight aortic and coronary, moderate cerebral.
Infarct of pons.

Pathologic Discussion

Dr. Tracy B. Mallory: This patient entered this hospital only near the end of a protracted illness, in a condition which made a complete work-up impossible. The history was pieced together from the scanty hospital records and the notes of one of the consultants who saw her from time to time. Unfortunately it fails to give an adequate picture of what was undoubtedly a difficult diagnostic problem in the early stages even though it became a very obvious one in the terminal period.

When she first sought medical attention with a history of only mild diarrhea, occasional traces of blood in the stools and a marked anemia, it was a real question whether she was suffering from dietary deficiency with secondary bowel pathology or whether a primary bowel lesion was producing a secondary deficiency syndrome. Both combinations have been shown to be possible and in fact not infrequent. Several competent examiners felt that the blood smear at that time was strongly suggestive of pernicious anemia and a trial period of high vitamin and liver therapy was not unreasonable. Even if the diagnosis should eventually prove to be ulcerative colitis the case appeared to be sufficiently mild so that no great risk was involved in delaying ileostomy for a short period.

In the other two cases we have presented today we showed two extremes of ulcerative colitis—one dying in a few weeks from onset, the other persisting without very disabling symptoms for eight years until finally a superimposed carcinoma caused death. In cases of the first type, ileostomy can probably never be done too soon and in the latter type it may be definitely postponed. The case under discussion might fairly be considered as a combination of the two types, in which a sudden exacerbation of a mild lesion almost overnight shifted the picture to that of the fulminant type. It is the danger of such an exacerbation, often coming when it is least expected, that makes these cases so difficult to handle. In retrospect it is easy to say that ileostomy was postponed too long, but I doubt if any of us would have wished to urge it strongly upon a reluctant patient in the early stages of the disease.

The final terminal event, I have never seen before in a case of ulcerative colitis. It was an acute mesenteric thrombosis which involved the entire small bowel. The terminal hemorrhage was from infarcted small bowel, not from the colon, which showed a rather average grade of ulcerative colitis with shallow denudation of the mucosa everywhere but no deep ulcers that were in danger of perforating or producing hemorrhage. If this mesenteric thrombosis had not supervened, the ileostomy would have been done in plenty of time.

Dr. Bock: Why did she have mesenteric thrombosis?

Dr. Mallory: I cannot answer that. Anyone with infection anywhere in the bowel has a perfect right to have a spread of infection into the veins draining the bowels; but it certainly is very unusual.

Dr. Bock: Why do we not see more of it in cases of ulcerative colitis then?

Dr. Mallory: I do not know. We practically never see it.

CASE 21072

Presentation of Case

A twenty-seven year old single Canadian nurse entered complaining of diarrhea of five weeks' duration.

Five weeks before entry the patient began to have diarrhea characterized by six movements a day. At the onset she had no chills, fever, nausea or vomiting. She did not feel particularly sick for the first three weeks. Although she was not working at that time, she felt tired and run-down and began to take yeast. Two weeks before entry she took a position in a store, but after working there awhile the diarrhea increased in severity. She developed fever and one evening had chills. She became nauseated but did not vomit. Her stools were never bloody but did contain a great deal of mucus. Five days before entry here she entered another hospital. She did not improve and had about seventeen bowel movements a day associated with much tenesmus. She continued to be nauseated and had anorexia. She also had generalized dull abdominal pain. Two weeks before entry she had difficulty in starting urination. This condition continued for one week. However, she had no dysuria. Eight months before entry, following a nervous breakdown which had kept her in bed for four months, she had arthritis which shifted from one joint to another without redness or swelling. This included the sternoclavicular

and temporomandibular joints. No specific treatment was given. She was in bed for five months.

Her family history and the rest of her past history are non-contributory.

Physical examination showed a well-developed and nourished, acutely ill woman. Her face was flushed. The heart and lungs were negative. The abdomen was moderately distended and showed increased tympany. There was no spasm, but there was increased peristalsis and tenderness in the right lower quadrant. The spleen was not felt. The blood pressure was 130/60.

The temperature was 103.4°, the pulse 112. The respirations were 24.

Examination of the urine showed a specific gravity of 1.020, 4 to 50 white blood cells, 2 red blood cells and numerous bacteria. The blood showed a red cell count of 3,930,000, with a hemoglobin of 60 per cent. The white cell count was 10,800, 71 per cent polymorphonuclears. Of thirty-five stool examinations fourteen showed positive guaiac tests. No amebae or pathogenic organisms were cultured.

Her temperature remained elevated. On the fifth day a proctoscopy showed in the small area examined a slightly edematous, granular mucous membrane, without ulceration. The mucus was increased in amount. There was, however, a 4 or 5 millimeter punched-out spot from which creamy pus exuded. Two days later moist râles were heard at both bases, associated with slight cough. Edema of her feet developed. On that same day an ileostomy was performed. Her condition remained about the same immediately following the operation, but on the second postoperative day her temperature began to rise, reaching 104°. Her abdomen remained distended. She was given intravenous fluids. The pulse rose to 160 and she went progressively downhill and died that day.

Differential Diagnosis

Dr. E. Parker Hayden: It seems to me that the history is pretty definitely that of an ulcerative colitis. It certainly is not a history of malignant disease, with the degree of fever which she had. As to just what happened at the last, one can only speculate.

Her discomfort was a little suggestive of obstructive pain, and the ileostomy could have been done for obstruction. It is much more likely, however, that she was diagnosed as a fulminating case of ulcerative colitis, resembling a very sick typhoid; and the ileostomy was done in an effort to head off the disease and possibly save her life.

I think it is quite possible that she may have had a little perforation which did not at first give her a general peritonitis but might have accounted for the distention, a slow perforation. Death may have been due to subsequent spread of infection, or may have been due to peritonitis originating from the ileostomy, either from the technical standpoint, or possibly due to the necessary handling of the bowel in doing the ileostomy. I should think that the history pointed pretty definitely to an acute ulcerative colitis and terminal peritonitis.

Dr. James H. Townsend: I do not think that this story brings out the events immediately preceding her death. She did pretty well for two days after operation although she was toxic and had high fever and rapid pulse. Shortly after midnight on the day she died she rather suddenly became much worse. I saw her at about half past three that morning. When I came in the change in her condition was very striking. She was very much paler than she had been before. When I went to feel the abdomen I found the bed full of bright red blood, passed by rectum. There must have been at least two quarts in the bed. We started to get everything ready for a transfusion but she died before it could be arranged.

I have nothing else to add except that she was exceedingly sick from the very beginning and all the specific things seemed to be ruled out, such as typhoid, bacillary, and amebic dysentery. She had an acute fulminating form of ulcerative colitis. The immediate cause of her death was hemorrhage.

Dr. Thomas V. Urmy: I think that she represents one of the relatively small group of very acute and severe colitis cases which in the great majority here have terminated fatally. In almost all of the cases the operation had been postponed too long and perforation with peritonitis had already occurred. We suspected it in this patient, even before ileostomy, because of the distention.

I remember one of these cases that came in near the onset, had ileostomy done at about two weeks, and did recover after a tremendously stormy period.

Clinical Diagnoses

Acute ulcerative colitis.
Hemorrhage from colon.
Nutritional edema.
Bronchopneumonia.

Dr. E. Parker Hayden's Diagnoses

Acute ulcerative colitis.
Peritonitis.

Anatomic Diagnoses

Acute ulcerative colitis.
Hemorrhage into large intestine.
Operative wound: ileostomy.
Bronchopneumonia, right lower lobe.
Anemia, secondary.

Pleuritis, chronic, left apex.
Leiomyomata uteri.
Operative scar: appendectomy.

Pathologic Discussion

Dr. Tracy B. Mallory: This case, as Dr. Urmy said, fits in the group of fulminant ulcerative colitis. We have had a considerable number of them and the mortality rate has been extremely high.

The cause of death was apparently primarily an acute hemorrhage from the bowel. The entire large intestine was found completely filled with bright red blood. The gut itself showed innumerable deep, sharply pitted ulcerations throughout its extent, rather more numerous in the cecum or ascending colon than in the descending colon and sigmoid, where we ordinarily expect to find them most numerous.

The peritoneal cavity was free from fluid, showed no evidence of peritonitis; but in attempting to remove the bowel wherever we touched it practically fell apart. I think there were no antemortem perforations but we made a dozen before we succeeded in getting the colon free from the mesentery. The difficulties that a surgeon is up against, of course, are even greater, and the most delicate handling of the bowel may easily produce perforation at the time of operation.

As Dr. Urmy suggests, I think the only possible chance is an immediate ileostomy. The difficulty is in recognizing them as cases of the fulminating type early enough to do the ileostomy in time.

Dr. Richard H. Miller: For those of you who later on are going to do surgery, it is well to remember that you must never explore and handle the colon in a case of this sort because not only can you tear it but the simple handling of it may give rise to infection. Dr. Daniel F. Jones told me something once which I have always remembered. He compared the colon in a case of this sort to the inside of a canvas tent. When it is raining very hard you can stand inside and the inner surface of canvas is dry, but if you run your fingers over it water comes promptly through and drips down. He always said that if you handle the bowel you can injure it just enough so that bacteria might penetrate and you must never handle the large intestine under these conditions.

A Physician: Where did the bleeding come from?

Dr. Mallory: From one of these ulcers, undoubtedly; we were not able to demonstrate exactly which one.

A Physician: How often do you see these patients die from hemorrhage?

Dr. Mallory: It has been much less common than perforation. In my experience there are ten perforations for one hemorrhage.

She had a very small amount of bronchopneumonia, but I am sure that the hemorrhage was the main factor.

Dr. George W. Holmes: One might emphasize again the danger of giving barium enemas to these patients when the bowel is in that condition.

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MEDICAL BIOGRAPHY

BIOGRAPHY is by all tokens one of the most subtle and difficult of the literary arts, the more so that it is so easily simulated by mere history. True biography is much more than a clear, dispassionate, and veracious record of the facts and achievements of a man's life. It differs from history as portraiture differs from photography. It should aim not merely to preserve the data and experiences of an individual existence, but from this factual background to evoke the illusion of personality, to recreate the magic of vitality.

Success in this aim is more difficult and more rare than in the field of fiction. For the creator of fictitious character is unhampered by fact and needs only to make his portrait true to the fundamental verities of nature. The creature of his imagination breathes with the spirit which he infuses, but need not correspond exactly with so complex a piece of work as a human being that has actually existed. The real biographer must recapture the spirit that has fled, provoke from the past the form and moving of the present, convert the mystery of death into that of life, make his printed pages speak like the colors of a canvas, restore and immortalize the eternal significance of a human personality.

For these reasons the number of true biographers in medicine is as few as in every other field of mortal activity; nay fewer, for scarce any but a physician is capable of appreciating and recapturing the inner problems and significance of a doctor's life and work, and few physicians have the time and gift for such pursuit. Among modern medical biographers one can single in a moment the few that attain measurably to the ideal. The personality and the immeasurable significance of Pasteur live for us forever in the pages of Vallery-Radot. Sir Rickman Godlee has masterfully preserved for all time the massive and dominating reality of Lord Lister. How express and admirable is the portrait of Sir William Osler by Dr. Harvey Cushing, whose sympathetic perspicacity and literary acumen adapted him perfectly to catch and recreate the charm and humanity of his subject!

Lately there has appeared another of these rare phenomena which seems to deserve inclusion in our group of really great medical biographies*. Franklin Paine Mall was little known to the general public or even to the rank and file of the medical profession. His work had not the immediate human contacts of that of Pasteur, Lister, and Osler, nor was his retiring character such as to give him overpowering dominance and prominence in his day. But his influence upon medical science and education, and upon those fortunate with whom he did come in contact, was so great and far-reaching as to make him one of the leaders of his generation. Dr. Sabin was one of his pupils and coworkers for twenty years, and the theory of education revealed in her volume is the very essence of his contribution to medical and human progress. Present and future generations of students, who knew him not, may now learn through these pages how he has influenced their lives, and gain an insight into the difficulties through which his generation won the privileges which they enjoy.

Particularly in Dr. Sabin's portrayal of Mall should praise be given and attention directed to the sixth and seventh chapters, which are the center, heart, and masterpiece of her achievement. In these chapters is presented the development of the inductive method of teaching, which was the fruition of his life and work; in them is the living revelation of the soul and genius of his personality as teacher-investigator. Teachers and students of the present and of the future may well take these chapters, and the ideas and example of Mall which they portray,

*Franklin Paine Mall. By Florence Rena Sabin. Baltimore, Md., The Johns Hopkins Press, 1934.

as the gospel, law, and creed of scientific education.

Though not conspicuous in the general eye, Mall was appreciated even in his own day, and has become increasingly recognized, as a very great man. Dr. Sabin's biography, which she truly entitles "The Story of a Mind," preserves indelibly the living personality of his greatness.

> "For not in wealth or popular acclaim,
> Not in the tribute and the praise of men,
> But in devout nobility of thought,
> In single and unselfish purposes,
> In great deeds greatly dared and truly done
> In silence and in pain, lies human triumph."

THE FINANCIAL BURDEN OF TUBERCULOSIS

A RECENT study of tuberculosis as found in the Bellevue-Yorkville district of Manhattan, in Hudson County, Maplewood and the Oranges, New Jersey, and Fulton County, New York, by Ruth Abelson Seder of the National Tuberculosis Association indicates that the total cost to the 650,000 Americans suffering with the disease is about $350,000,000 a year based on the average duration of the disease of five years. This means according to the report as published in the *New York Times* a cost of $2,000,000,000 for the five years of invalidism of this group.

The most important feature of this tremendous burden is that it kills more people in early adult and up to middle life than any other disease and hence is a loss to the country beyond our power to evaluate.

The estimates set forth find a definite support in the records of new cases reported in the United States in 1933, for this number is 111,844 without the records of Arkansas, Idaho and North Dakota, and that for every new case reported there may be five others neither recognized nor discovered.

The agencies employed in the contest against tuberculosis are the official organizations of the Federal Government, the States and municipalities endorsed and augmented by the two thousand voluntary organizations.

As has been recognized and often emphasized, the early discovery of the disease is the most important factor in reducing the cost of tuberculosis and the mitigation of the suffering incident thereto.

Furthermore the possibility of reducing the cost imposed by the malady and the salvaging of life of the victims depend more on early treatment than any other or indeed all other factors, for appropriate treatment within six months of the first symptoms reduced the average expense by about $250 per patient. If, on the other hand, delay exceeds one year, the average cost of this group exceeds thirty-nine hundred dollars per average patient which is nine hundred dollars above that of the average cost of treatment.

It is the consensus that sanatorium treatment is essential in this disease and that the average cost is $3.50 per day. Patients in the incipient stage average to require 5.6 months' stay in the sanatorium while those of the second stage usually remain 5.9 months and those in the third stage 9.6 months. The saving to the patient or the public of the difference of four months of institutional treatment, as shown in the records of the different stages of the disease, presents a problem of great economic importance so far as the financial burden applies, but in addition the many homes disrupted by the illness of the wage-earner add to the individual and community problem.

Taken by and large this disease warrants far more attention than has been given to it and more definite measures for its control are indicated. Whether the public health policies of the nation will have to be made more effective will depend upon the response of the medical profession to the requirements of the situation. Even with all of the advances made and underway, a great number of these afflicted people are infected and neglected because of ignorance and inadequate medical attention. Public instruction must continue in this field and be even more definite.

The average doctor usually sees the patient first and the question is pertinent as to whether he has been sufficiently well trained to suspect the disease in its incipiency or whether he is on the lookout for this disease in the so-called run-down patient. The doctor who can and will detect tuberculosis early and arrange for appropriate treatment immediately is in the best strategic position for attacking the disease. If there are too few such men, the complaint of some of our phthisiologists warrants consideration by the medical schools.

The Massachusetts Medical Society

THE SHATTUCK LECTURE

"Sprains and Dislocations" will be the subject of the Shattuck Lecture to be delivered by Dr. W. E. Gallie of Toronto, Canada, June 3, as a feature of the Annual Meeting.

William E. Gallie, M.D., F.A.C.S., F.R.C.S., (Eng.), University of Toronto Faculty of Medicine 1903. Professor of Surgery, University of Toronto Faculty of Medicine. Surgeon-in-Chief, Toronto General Hospital.

Photograph by Bachrach

THOMAS JAMES O'BRIEN

DR. Thomas James O'Brien died on February 5, 1935 after an illness of a little less than four months.

He was born in Cambridge, July 12, 1872, the son of Edmond and Ellen M. O'Brien.

In 1895 he was graduated from the Massachusetts College of Pharmacy and in 1899 received the degree of Doctor of Medicine, cum laude, from the Harvard Medical School. Following an interneship at the Carney Hospital for a year he became assistant resident physician at the Long Island Hospital. From 1900 to 1907 he was out-patient physician at the Carney Hospital.

Dr. O'Brien married Miss Margaret Anne Learson in 1903 who died in December 1909 leaving three sons and a daughter to be educated and trained by their father. For twenty-five years he bore unaided the triple responsibility of his growing practice, the careful superintendence of his children's lives, and the management of his household. To these were added his duties on the staff of the Boston City Hospital, his professorship at Tufts, and his contribution to the executive work of the Massachusetts Medical Society.

His children all received excellent educations, three of them being established in life and one soon to graduate from a professional school.

At the Boston City Hospital and the Tufts College Medical School, his talents, his skill, his studious attention to detail and his charming personality attracted many of the graduates who found in him an ideal consultant.

He was a former vice-president of the Massachusetts Medical Society but it was in the position of assistant to the president that he became well known throughout the state.

His work was so satisfactory that he was reappointed by several presidents and under his direction the business of the headquarters office was efficiently managed. Long experience made him a valuable aid to the president and he gave unsparingly of his time and thought. He accompanied the president on his frequent visits to district society meetings where he was always received with enthusiasm, often with affection. As a member of the Committee on State and National Legislation he carefully catalogued the bills and gathered our forces at the legislative hearings.

Only a few months after his retirement from the hospital and medical school and while he was planning to give his whole time to practice and the society, he was stricken with a fatal disease. He examined his x-ray plates and quickly arrived at the correct conclusion and accepted his fate with amazing fortitude.

In addition to Dr. O'Brien's professional and executive attainments, he was a gentleman in the truest sense of the word, loyal, courteous, modest, dependable.

In his passing the Massachusetts Medical Society has lost an officer whose services were of inestimable value; and his friends, a staunch, sympathetic and delightful companion.

THIS WEEK'S ISSUE

CONTAINS articles by the following named authors:

MILLER, RICHARD H. A.B., M.D. Harvard University Medical School 1910. F.A.C.S. Assistant Professor of Surgery, Harvard Medical School; Associate Surgeon, Massachusetts General Hospital. His subject is "Acute Hematogenous Osteomyelitis." Page 283. Address: 264 Beacon Street, Boston, Massachusetts.

MARBLE, ALEXANDER. M.D. Harvard University Medical School 1927. Physician, New England Deaconess Hospital. Assistant in Medicine, Harvard Medical School. Address: 81 Bay State Road, Boston. Associated with him are

ROOT, HOWARD F. M.D. Harvard University Medical School 1919. Physician, New England Deaconess Hospital. Assistant in Medicine, Courses for Graduates, Harvard Medical School. Address: 81 Bay State Road, Boston. And

WHITE, PRISCILLA. M.D. Tufts College Medical School 1923. Physician, New England Deaconess Hospital. Instructor in Pediatrics, Tufts College Medical School. Address: 81 Bay State Road, Boston. Their subject is "Diabetic Coma." Page 288.

OLIVER, E. LAWRENCE. M.D. Harvard University Medical School 1904. Clinical Professor of Dermatology and Syphilology, Harvard Medical School. Chief, Department of Dermatology and Syphilis, Massachusetts General Hospital. Dermatologist, Children's Hospital and Faulkner Hospital. Consulting Dermatologist, Massachusetts Eye and Ear Infirmary, Peter Bent Brigham Hospital, Long Island Hospital (Boston) and Cable Memorial Hospital, Ipswich, Mass. Consultant, Beth Israel Hospital and Elliot Community Hospital, Keene, N. H. Address: 20 Fairfield Street, Boston, Mass. Associated with him is

CRAWFORD, G. MARSHALL. B.M., M.D. Northwestern University Medical School 1932. Assistant, Department of Dermatology and Syphilology, Massachusetts General Hospital and Harvard Medical School. Address: 416 Marlborough Street, Boston, Mass. Their subject is "Iodo-Bismuthate of Quinine in the Treatment of Syphilis." Page 297.

WASHBURN, FRANK H. M.D. Tufts College Medical School 1899. F.A.C.S. Surgeon and Chief of Staff, Holden District Hospital, Holden, Mass. Thoracic Surgeon, Worcester County Sanatorium, Worcester, Mass. Consulting Surgeon, Rutland State Sanatorium, Rutland, Mass. and Harrington Memorial Hospital, Southbridge, Mass. His subject is "What We Have Learned From One Hundred Intra-Pleural Pneumolyses." Page 300. Address: Holden Clinic, Holden, Mass.

The Massachusetts Medical Society

SECTION OF OBSTETRICS AND GYNECOLOGY*

THOMAS ALMY, M.D., *Chairman*, 140 Rock Street, Fall River, Mass.

C. J. KICKHAM, M.D., *Secretary*, 524 Commonwealth Avenue, Boston, Mass.

ROUTINE FOR DETERMINATION OF PELVIC MEASUREMENTS IN PREGNANT PATIENTS

WHILE we have all met with cases presenting marked dystocia that pelvimetry has not warned us of, nevertheless, pelvimetry is an indispensable adjunct and guide in the diagnosing of abnormal pelves, and it behooves each and every one who practices obstetrics to perform it systematically. There are two types: viz., External and Internal Pelvimetry.

EXTERNAL PELVIMETRY: Our routine is as follows: The gravida is placed in a dorsal position on the examining table. We place the tips of the pelvimeter on the anterior superior spines of the ilia and count in centimeters the distance between the two spines (interspinous diameter). We then measure in like manner the distance between the iliac crests (intercrestal diameter). We now have the patient turn on her left side and with one tip of the pelvimeter at the left sacroiliac synchondrosis we measure to the tip of the right anterior superior iliac spine (left oblique diameter). Now turning the patient on her right side, we measure in like manner from the right sacroiliac spine to the tip of the left anterior superior iliac spine (right oblique diameter). Now from the depression under the last lumbar spine we measure to the anterior surface of the pubis;—this is the external conjugate or Baudelocque's diameter. This lozenge-shaped space, marked by the four dimples and which the Greek sculptor pictures so clearly in his Venus de Milo, is called the Rhomboid of Michaelis. The external conjugate is measured from the upper dimple. Interesting to note is that this rhomboid is distorted in manner with contracted pelves, squat in flat pelves, and irregular if the women are asymmetrically built. This latter would also be evidenced by abnormal external conjugates.

INTERNAL PELVIMETRY: The patient is now placed in the lithotomy position. If she is in the early stages of gestation, strict asepsis need not be carried out. The external genitalia are cleansed with a weak bichloride solution. With clean boiled gloves the labia are separated with the index finger and thumb of the right hand. Downward depression of the perineum is now

*A series of short selected articles by members of the Section will be published weekly.
Comments and questions by subscribers are solicited and will be discussed by members of the Section.

made with the middle finger of the left hand and the index finger of the same hand is gently inserted into the vagina, thus avoiding any contamination with the external genitals. A careful inspection is now made for evidences of infection about the outlet, the presence or absence of cystocele or rectocele. The cervix is gently palpated for its softness, parity and presence or absence of lacerations or erosions. Now contact the tip of the middle finger on the promontory of the sacrum, by passing the examining fingers underneath the cervix. Then place the tip of the right index finger against that part of the left index finger that impinges against the inferior border of the pubis, remove the hand, and measure this distance with our pelvimeter. This is our diagonal conjugate. To obtain the conjugate vera, we deduct one and one half to two centimeters. While this procedure is being carried out, the pubic arch should be palpated for its shape and general contour, also the uterus for its size, mobility and position, both vaginal vaults for any pathology, thus completing the bi-manual examination. Now with the tips of our pelvimeter against the tuberosities of the ischia, we measure this distance which gives us the transverse diameter of the outlet or the breadth of the canal.

MASSACHUSETTS LEGISLATIVE NOTES

The Hearing on Bills for and against Vaccination will be conducted before the Committee on Public Health, February 19, at 10:30 A.M.

That introduced by the Commissioner of Public Health, extending the requirements to private schools, is the same as the one Dr. S. B. Woodward has valiantly championed for many years. This is important and has failed of favorable action because of the indisposition of doctors to be present in sufficient numbers to impress members of the Legislature.

The other bills on vaccination are designed to defeat that introduced by Commissioner Chadwick.

S. 143. Resolve providing for an investigation by the Department of Public Health relative to the discharge of sewage into Boston Harbor with a view to the elimination of objectionable conditions in the vicinity of said Harbor.

EDITORIAL NOTE: This seems reasonable unless the Department of Public Health already has adequate data.

AN ENDORSEMENT OF JUSTICE TO HOSPITALS AND DOCTORS

The Barnstable District Medical Society has sent a letter to the Joint Judiciary Committee, Massachusetts Legislature, endorsing the principles of justice and fair play to hospitals and to the medical profession as embodied in Senate bill number 52.

MISCELLANY

THE APPOINTMENT OF DR. LOUIS A. O. GODDU

Dr. Louis A. O. Goddu has recently been appointed by the Board of Managers of The Boston Dispensary to the position of Associate Chief in the Orthopedic Department of that institution.

TEACHER COÖPERATION IN A CLINIC PROGRAM

BY BERNICE M. HENDERSON

Educational Secretary, Massachusetts Society for Mental Hygiene

With the increasing emphasis upon the mental health of the school child there is need for a better mutual understanding between school and psychiatric clinic, a clearer appreciation of the functioning of each organization. Throughout the state of Massachusetts there are a variety of clinics designed primarily to serve the needs of the school child. These are conducted by the Division of Mental Hygiene, the out-patient departments of some of the state hospitals, a few general hospitals, and still others under private direction. As these clinics (with the exception of certain provisions in some localities for the examination of retarded children) are not operated as an integral part of the school system, there exists among many parents a resistance to their use which is not generally found in the case of similar clinics coming directly under the administration of the school department. This resistance must be overcome, and mainly through the efforts of the teacher. The purpose of the clinic, which is primarily to aid in the more satisfactory adjustment of the individual child, cannot be successfully carried out without the coöperation of parent and teacher. To a large extent the confidence which the average parent has in such a clinic is a reflection of that evidenced by the teacher. It is therefore essential that the teacher believe in the work of the clinic, and in order to believe in it she must understand its purpose and functioning.

In our efforts to understand the child it seems urgent, in these days of all sorts of exploitation of psychological theories, to distinguish in so far as possible between well-founded and scientific theories of behavior and those frequently more popular but less scientifically established. Here again we look to the teacher as a professional person, capable of understanding the principles under which a child guidance program operates, and able to help interpret such principles to parents, who in many instances have not had equal opportunity to gain such understanding.

Unfortunately there has long been prevalent an idea that the child guidance clinic deals mainly with the so-called "problem" child, with the result that the average parent tends immediately to become on the defensive when a teacher suggests she take her child to a clinic. If the teacher wishes the help of the

specially trained personnel of the clinic, she must herself understand that while the clinic does treat more serious situations, its greater emphasis is in the field of prevention, its interest is in helping in the better adjustment of the so-called "normal" child who, because of poor habit training, physical condition, or special emotional make-up, may need special handling. It aims to help the average child meet the problems incidental to certain phases of his development. Teachers recognizing these aims of the clinic may find help for the everyday problems arising in the classroom by having a better understanding of the special reactions of children of special make-up.

The clinic depends upon the teacher for data and observations of diagnostic import and for assistance in carrying out recommendations with observations which tend to check those made in the clinic. It also depends upon her for the further interpretation of those suggestions which it makes to the family. To the parent the clinic represents a more or less unknown quantity. The teacher's attitude can do much to overcome hesitancy on the part of parents to coöperate, especially when the clinic makes recommendations which are in opposition to the parents' wishes or preconceived ideas of what the situation demands.

There have been certain criticisms of these clinics on the part of teachers, namely, that the clinic will accept only certain cases, that it takes too long to accomplish results, that impractical suggestions are brought back, or that "nothing happens," or that the clinic expects the teacher to devote a disproportionate share of her time to an individual child. To some extent these criticisms are justified in individual cases, but in the main are explainable in the light of reason. Limitation of intake is essential with limited clinic staff and selection of cases is also essential, or the teacher who complains that nothing happens may be justified in her complaint. While still in the process of proving its effectiveness, the clinic must select, in the main, cases where there may be demonstrable results, where there is reasonable assurance that the environment can be manipulated to meet certain needs of the child, and where the child himself is capable of changing to meet certain demands of his social milieu. Psychiatrists are not infallible, and teachers should feel free to indicate wherein the situation seems not to bear out their observations.

The teacher, on her part, must be very careful that she treats confidential data provided for her use in the child's behalf as such, and not as something to be passed on in conversation with others. She must never use the clinic as a disciplinary agent. The clinic staff in its turn must respect the opinion and observations of the teacher, with due regard for restrictions of time and administration policies, asking of her only such help as she may reasonably be expected to carry out.—*Massachusetts Society for Mental Hygiene.*

MAKE MATERNITY SAFE

A nation-wide campaign to make maternity safe has been planned by the Maternity Center Association, 1 East 57th Street, New York City. The climax of the effort will be timed for the week previous to Mother's Day, which falls this year on May 12.

Mrs. Shepard Krech, President of the Association, has sent out a widespread call for the coöperation of churches, women's clubs, civic and educational institutions, offering without charge to supply groups with suggestions for local programs to improve maternity care.

"Our object," states Mrs. Krech, "is to make maternity safe. Leading authorities tell us that at least one-half the deaths in childbirth could be prevented by adequate maternity care. More than 15,000 women in this country die annually in childbirth. The death rate has remained practically stationary during the last twenty years, despite the knowledge which exists and is available in almost every community in the country, which could save these women.

"Not until the average parent realizes that the mother of to-day is not getting the care that makes motherhood safe, will this situation be changed. It will not be changed so long as fathers believe they have discharged their entire duty toward motherhood by sentimental expressions on Mother's Day, forgetting that there are 365 mother's days in every year, and that on each of them women have babies.

"To-day safe maternity care is not available to every expectant mother at a price she can afford to pay. Tomorrow it can be, if lay and professional people in every community will combine in the effort to inform parents everywhere what the six principles of adequate maternity care are, so they can demand that this care be made available in every community, rural or urban.

1. A complete medical examination early in pregnancy.
2. Regular and frequent medical supervision.
3. An aseptic delivery under the supervision of an obstetrician.
4. Supervision, care and instruction until the mother is able to resume her work.
5. Examination of the mother at six weeks, three months, six months and one year after the baby is born.
6. Arrangements for continuous medical supervision of the baby.

"Parents and doctors working together with community organizations should be able to secure complete maternity care for every expectant mother in the community whether she goes to a private doctor or to a hospital clinic.

"Do not allow another Mother's Day to pass without taking the first step toward making maternity safe for the mothers in your community."

CORRESPONDENCE

RESTRICTION OF ATTENDANCE AT THE INTERNATIONAL POSTGRADUATE COURSES

February 2, 1935.

Editor, *New England Journal of Medicine*,

On page 220 of The *New England Journal of Medicine* of January 31, 1935, there is an announcement regarding "the International Postgraduate Courses that have been arranged for medical postgraduate training under the auspices of the Lord Mayor of the City of Berlin, etc." The announcement in the *Journal* ends with the following: "German as well as foreign doctors may attend the course."

One very important item has been unknowingly omitted in your *Journal* and that is that non-Aryan citizens (physicians) of the reich are not admitted to the courses. The last-named exception is made plain in the A. M. A. foreign letter from Berlin dated November 19, 1934 (*J. A. M. A.*, 104:331 [Jan. 26] 1935). Non-Aryan citizens (physicians) outside of Germany intending to attend the above courses should have the specific requirements for attendance placed before them so that they will steer clear of where they are not wanted.

Sincerely yours,

J. L. Grund, M.D.

483 Beacon Street, Boston, Mass.

PROPHYLACTIC FORCEPS

February 4, 1935.

Editor, *New England Journal of Medicine*,

I am glad you have resumed the selected articles by members of the Section of Obstetrics and Gynecology. I think all of us have found them interesting and instructive.

I would like to say, however, that I am not wholly in accord with the first in the new series, "Is the Prophylactic Forceps Procedure Justifiable?" I have had a moderate obstetrical experience incidental to twenty-five years of general practice, a large part of which has been in a suburban hospital of eighty beds. I have also assisted my fellow practitioners many times as anesthetist and fundus guard. I have done prophylactic low forceps many times and I have also been present when some of my local colleagues have been doing the same operation. It is my opinion that the majority of general practitioners take operative obstetrics seriously, know the potential danger of every instrumental delivery, know the importance of having the head well down on the pelvic floor, the importance of having the head fully rotated, and also know how to repair an episiotomy wound. I think a large majority know the importance of a careful examination to determine whether the head is fully rotated and if not, know how to change a transverse arrest to an anterior position. None of us like to deliver babies whose eyes are gazing at the ceiling. Is the repair of an episiotomy wound such a formidable surgical procedure?

For years I have used a guide suture after the incision which makes possible an early and accurate approximation of the divided structures. Doubtless other practitioners have done the same. The only reference to this simple but helpful procedure was in a recent paper in The *New England Journal of Medicine* by Dr. Benjamin Burpee of Manchester, New Hampshire.

At a meeting of a British Obstetrical Society some time ago the remark was made that the maternal and infant mortality might be lessened if all cases were referred to the midwives who had very good records. This high mortality among the profession was said to be due to meddlesome obstetrics. Who is it that institutes meddlesome obstetrics? Not the general practitioners, some of whom are said to have excellent records seldom using forceps. Could the tendency to make labor an elective procedure by early rupture of the membranes be called meddlesome obstetrics? I am not prepared to discuss the advantage or disadvantage of such a procedure but can refer to Dr. DeLee who has a convincing argument against it.

I do not believe we know the final word in regard to the value of the barbiturates in the conduct of a labor. It reads well in the *Ladies' Home Journal* and sounds well in an address by an obstetrician before a mothers' club, but all of us know that its practical application has its difficulties.

I recently heard over the radio a statement that with our present knowledge every woman was entitled to a painless labor. This seems to me to cover a lot of territory and conditions.

This communication does not mean that I am not deeply appreciative of the good counsel I have had from obstetrical consultants in the past.

Yours sincerely,

Alfred A. Fenton, M.D.

471 Washington Street,
Norwood, Mass.

ARTICLES ACCEPTED BY THE AMERICAN MEDICAL ASSOCIATION COUNCIL ON PHARMACY AND CHEMISTRY

535 North Dearborn Street, Chicago, Ill.,
February 4, 1935.

Managing Editor,
The *New England Journal of Medicine*,

In addition to the articles enumerated in our letter of January 3 the following have been accepted:

Bilhuber-Knoll Corporation

Dilaudid Compound Tablets ½ grain

Hoffmann-La Roche, Inc.

Alurate

Alurate Tablets, 1 grain

Elixir Alurate

Sodium Alurate

Capsules Sodium Alurate, 3½ grains

Jensen-Salsbery Laboratories, Inc.

Diphtheria Toxoid, Alum Precipitated (Refined)

Merck & Co., Inc.
Cebione—Merck
Ampules Cebione—Merck, 0.1 Gm.
Tablets Cebione—Merck, 0.01 Gm.

Wm. S. Merrell Co.
Diothane Ointment 1% Ophthalmic Tube

Frederick Stearns & Co.
Neo-Synephrin Hydrochloride Jelly

Winthrop Chemical Co., Inc.
Ampules 1% Isotonic Solution Tutocain with Suprarenin, 1:20,000, 3 cc.
Tablets Alypin 1/3 grain

Nonproprietary Articles
Cevitamic Acid
Aminoacetic Acid

The following product has been accepted for inclusion in the List of Articles and Brands Accepted by the Council But Not Described in N.N.R. (New and Nonofficial Remedies, 1934, p. 443):

Merck & Co., Inc.
Creosote—Merck

Yours sincerely,
PAUL NICHOLAS LEECH, *Secretary*,
Council on Pharmacy and Chemistry.

RECENT DEATHS

O'BRIEN—THOMAS JAMES O'BRIEN, M.D., for many years a practicing physician in Boston, died at his home, 501 Beacon Street, Boston, Tuesday, February 5, 1935, after an illness of three months.

He was born in Cambridge, Mass., July 12, 1872, the son of Edmond and Ellen M. O'Brien, and received his early education in the Cambridge public schools. In 1895 he graduated from the Massachusetts College of Pharmacy, and in 1899 was graduated cum laude from Harvard Medical School. He then interned at the Carney Hospital, until July, 1900, when he became Assistant Resident Physician at the Long Island Hospital. He was Physician in the Out-Patient Department of the Carney Hospital from 1900 to 1907. From 1901 to 1905 he taught analytical and organic chemistry at the Massachusetts College of Pharmacy. He was a Trustee of this institution from 1907 to 1912, and in the year 1911-1912 was Dean. During his tenure of this office he was instrumental in securing a contribution from the George R. White Fund for the purpose of the erection of the present college building on Longwood Avenue.

He was a member of the Faculty of Tufts College Medical School from 1902. In 1920 he was appointed Professor of Clinical Medicine and served in that capacity until the time of his death. The Class of 1934, Tufts College Medical School, dedicated its yearbook, "The Caduceus," to him.

Dr. O'Brien was made Assistant Visiting Physician at the Boston City Hospital in 1914. In May, 1919, he became Secretary of the Medical Staff, which office he held until January, 1931. In January, 1932, he became a member of the Senior Staff, and in July, 1934, he resigned, having reached the compulsory age of retirement. At this time he received a letter from Dr. George G. Sears, Secretary of the Trustees, informing him that he had been appointed Consulting Physician, and expressing the appreciation of the Board for his services during twenty years. He was also a consulting physician at St. Elizabeth's Hospital, the Leonard Morse Hospital in Natick, the Marlboro Hospital, and many others in Massachusetts.

Dr. O'Brien was a former Vice-President of the Massachusetts Medical Society, and for the past few years had been Executive Assistant to the President. In connection with this office, he addressed many medical and educational organizations throughout the State, and frequently represented the Society at hearings on matters of medical legislation at the State House.

He was also Chairman of the Committee on Permanent Home, and a member of the Committee on State and National Legislation. He represented the Massachusetts Medical Society as a delegate to the Annual Congress of the American Medical Association on Medical Education, Licensure and Hospitals, at Chicago, from 1929 to 1934; and also in 1934 was the Massachusetts delegate to the Annual Meeting of the Secretaries of State Medical Societies. He was a former Censor of the Suffolk District Medical Society. He was the author of articles in The *Boston Medical and Surgical Journal* and The *New England Journal of Medicine*. He joined the Massachusetts Medical Society in 1900 and was also a Fellow of the American Medical Association. He held memberships in the American Heart Association, the New England Heart Association, the Massachusetts Society of Examining Physicians, the Boston Medical Library, and the Harvard Club of Boston.

Dr. O'Brien was married in Boston in 1903, to Margaret Anne Learson, who died in December, 1909. With him when he died were his four children: Mrs. Philip M. Dillon, of Albany, N. Y.; Edmond T., and Francis R., of Boston, and Richard, of Adams, Mass. Also surviving him are his sister, Mrs. Elizabeth Norris, of Belmont, and six grandchildren.

LIVINGSTON — ISABEL LIVINGSTON, M.D., of 247 South Street, Needham, Massachusetts, died at her home, February 4, 1935, aged eighty-two years.

Dr. Livingston graduated from the College of Physicians and Surgeons, Boston, in 1903. She was educated in the public schools of Needham, and, previous to her medical career, was head of the English department of the Lynn public schools, and, after graduating in medicine, was a member of the faculty of the College of Physicians and Surgeons, and on the visiting staff of the New England Hospital for Women.

She is survived by a brother, David H. Livingston, and four sisters, the Misses Elizabeth, Mary, Cora, and Janet Livingston, all of Needham.

REID — ROBERT ALEXANDER REID, M.D., of 36 Hyde Avenue, Newton, Massachusetts, died February 5, 1935, at his home. He was born in 1848 in Salisbury, Connecticut, the son of Rev. Adam Reid.

He graduated from the Bennett College of Eclectic Medicine and Surgery of Chicago in 1877, and settled in Newton about forty-five years ago, continuing practice until his retirement in 1920. He was editor of the *Massachusetts Medical Journal* for more than forty years before it ceased publication.

Dr. Reid is survived by three sons, Dr. William D. Reid, whose office is at 510 Commonwealth Avenue, Boston; George S. Reid, of Ipswich, Robert A. Reid, of West Acton; and a daughter, Mrs. William Stewart, of Hampden, Connecticut.

BAVUSO—ANTHONY CHRISTIANA BAVUSO, M.D., of 354 Hollis Avenue, Framingham, Massachusetts, died in that city, December 30, 1934. Dr. Bavuso was born in Boston in 1902, the son of Philip Bavuso and Catherine (Allegreti) Bavuso.

He graduated from the Middlesex College of Medicine and Surgery in 1927, and joined the Massachusetts Medical Society in 1934.

Dr. Bavuso was not married.

SULLIVAN — ANDREW JOSEPH SULLIVAN, M.D., of 27 Elm Avenue, Brockton, Massachusetts, died at his home, February 9, 1935. He was born in Brockton in 1885, the son of Cornelius A., and Margaret E. Sullivan, was educated in the schools of his native city and graduated in medicine from the Tufts College Medical School in 1909.

He joined the Massachusetts Medical Society in 1910. He was also a Fellow of the American Medical Association and the American College of Surgeons. He was a member of the New England Laryngological and Otological Societies.

He entered service in the World War at Portsmouth, New Hampshire Navy Yard, and was on the George Washington when troops were transported and also when President Wilson went abroad to attend the Versailles Peace Conference.

Dr. Sullivan was eye, ear and throat surgeon to the Goddard and Brockton Hospitals.

He is survived by five sisters, Mrs. John W. Condon, Miss Ellen M. Sullivan, Mrs. J. F. Lehan, Miss Elizabeth R. Sullivan, and Mrs. J. J. Welch, all of Brockton.

WAGNER — HARRY SAMUEL WAGNER, M.D., Superintendent of the Barnstable County Sanatorium at Pocasset, died at the Baker Memorial Hospital, February 9, 1935. He was born in Toledo, Ohio, in 1877, and educated at Dean Academy, and Green Springs Academy. He received his M.D. degree from the University of Michigan Medical School in 1903.

Dr. Wagner joined the Massachusetts Medical Society in 1919. After specializing in tuberculosis work, he accepted an appointment at the North Reading State Sanatorium in 1909 and in 1912 became Superintendent at the Hartford (Conn.) State Sanatorium. In 1915 he was appointed Superintendent of the State Sanatorium at Knoxville, Tennessee, and in 1919 became Superintendent of the Barnstable County Sanatorium.

His widow and three children survive him.

HOUGHTON—RICHARD H. HOUGHTON, M.D., of 308 Sumner Street, East Boston, died at the Peter Bent Brigham Hospital, February 8, 1935. Dr. Houghton was born in East Boston in 1880 and graduated from the Tufts College Medical School in 1905.

He served as school physician for the Adams School district, East Boston, and was president of the East Boston Medical Association and a member of the Knights of Columbus.

Dr. Houghton is survived by his widow, Mrs. Mary L. (Duane) Houghton.

BROWN—JAMES EDMUND BROWN, M.D., of Bryantville, Mass., and Providence, Rhode Island, died February 8, 1935. He was born in 1853 and graduated from the Baltimore College of Physicians and Surgeons in 1886.

He was a member of the Rhode Island Medical Society.

Dr. Brown is survived by a son, Sharon O. Brown, of Providence, Rhode Island, and a daughter, Mrs. Bernice Cronkhite.

OBITUARY

IN MEMORY OF DR. G. FORREST MARTIN*

DECEMBER 10, 1862-NOVEMBER 4, 1934

Two texts for this brief memorial have been selected as exemplified in the life of Dr. G. Forrest Martin, our lost friend.

(St. John 15-13.) "Greater love hath no man than this, that a man lay down his life for his friends."

(II Timothy 4-7.) "I have fought the good fight, I have finished my course, I have keep the faith."

The death of Dr. Martin in this city on November 4, 1934, in his seventy-second year, has taken from Lowell, one of her foremost private citizens, a man whose name, activities and broad human interests were known and praised throughout this Commonwealth, his adopted home of which he was most proud, and where he found so much happiness and joy, in living and in useful service to his fellowmen.

His was also a name of national significance, renowned through many states; throughout New England which he knew as from an open book; New York, where he studied and first practiced his profession; his native state of Ohio, steeped in the traditions of pioneers from this state of ours; the Atlantic States as far as Florida, where for impair-

*Adopted by the Middlesex North District Medical Society January 30, 1935.

ment of health he had to spend the winter months for many years. Everywhere he was acquainted with the leading men of the communities, in business, in learning, politics and trades, as well as in the medical profession; with the humble, the poor, and with those in affluence; whether man, woman or child; with whomsoever his comings, goings and tarryings, brought him in contact, he was ready to greet with a smile, to speak, to jest, extend his sympathy or lend encouragement; to ask for information or to give it on any topics of the day, or matters of importance, there was scarcely a subject he could not debate, nor field in which he had not exercised or plied his skill.

By wise and gentle parents, bred in a Christian home, the boy received his ethical and moral training, and was drilled in ways of thrift, honesty and truth, and grew up proudly relating his father's enterprises, his brothers' attainments and cherishing the beautiful likeness and teachings of his mother.

He was brought up to love his church, to industry in school and home, learning to do his chores, to serve, to work on farms, in cranberry meadows, and as he grew in stature, knowledge and in years, became a store clerk, kept the books or worked at draughting board on estimates in boiler works, taught, held singing schools and in many other ways plied his talents for the earning of his livelihood, and for his later schooling.

With the excellent training and the inspiration of teachers in the Lowell schools as a background, and with inborn diligence he later studied medicine at the New York Homeopathic Medical College and Flower Hospital, and never ceased to be a student until he had acquired proficiency in the science and art of medicine and surgery.

Dr. Martin was honored with appointments on many boards, commissions and societies. He was a member of the Staff of the Lowell General Hospital, the Board of Trustees, and Surgeon Emeritus. He had been a member of the Board of Trustees of the State Infirmary since 1920, and since 1926, the Chairman; a member of the State Advisory Committee for the control of cancer, and Chairman of the Cancer Commission of Lowell for eight years; a member and the Chairman of the Lowell Board of Health; a member and past President of the Massachusetts Homeopathic Society; member of the American Institute of Homeopathy, the Massachusetts Medical Society, the Massachusetts Surgical and Gynecological Society, the Middlesex North District Medical Society of which he was a past vice-president, and a Fellow of the American College of Surgeons.

He was a member of the Lowell School Committee, and served on the Lowell Public Safety Committee during the World War; he was a trustee of the Central Savings Bank, a member of the Lowell Chamber of Commerce; a most active member of the Grace Universalist Church, and an enthusiastic charter member, and a president of the Lions Club. He was widely known in Masonic bodies having reached the prominence of the 33rd degree in Masonry.

Dr. Martin was always so ready to respond or volunteer for service in good works for this city, that to his daily programs were added committee meetings and chairmanships for innumerable community interests, commemorations, hearings and welfare programs to an extent rarely equalled by others.

He was greatly interested in everything pertaining to construction and improvement in hospital buildings, equipment, organization, administration, and nursing schools. At the Lowell General Hospital and at the State Infirmary he was literally fighting for the interests of patients, nurses and for standards until the very last, and as he began to realize that his activities in the operating room would soon be past, he happily expressed himself as going to have just so much more time to devote to the greater hospital interests which were always in his thoughts.

Two years ago, after the critical attack of angina, from which he suffered in the mountains, when it did not seem as if he could come back to us again, it was a revelation by the second week to find his plans for returning, taking shape, for with his indomitable spirit he was using his enforced rest as a time for making further plans for these hospitals, and a revised program for his own busy days. He did come back and was fighting as hard as ever before; he pushed onward in his work and had the satisfaction of seeing many of his earlier dreams realized, and others nearing completion or beginning to take shape.

"He fought the good fight, he had finished his course, he had kept the faith," with his ideals. He had met with disaster, discouragements, defeats, griefs, sufferings and pain, but he never gave up fighting for his cause, and greater, were his triumphs, and his victories. Despite his warnings that the end could not be far away, at his work he could still be found, faithful, skillful, thorough.

The doctor found great pleasure in helping younger people, and especially younger physicians, and not a few have known, felt and cherished his kindly, fatherly advice, gained new inspiration and attained success, to which in no small degree they acknowledge his kindly and his timely aid.

In the home beside his loving wife he found his greatest joy, with every tender thought directed to her happiness; and when two younger generations gathered there, the rapture was complete.

His outstanding attribute seemed to be friendliness. He loved to have friends; his life was an expression of the sacred proverb, "A man that has friends must show himself friendly." He had friends everywhere, deep rooted in friendship due to kindly acts of his, unheralded and rarely known excepting to the possessors of his friendship.

His last illness was only of a week's duration and did not seem to indicate the end so near. Shortly before his passing, saying he was "feeling better,"

he began talking of the Princeton game, with his usual fervor, and to the last moment showed his interest in what was going on.

For us there remained only a passing look into the open casket where death had only slightly changed his countenance, fair, and soon to vanish from our sight.

The feelings that have been striving for expression are not ours alone, but are in common with unnumbered citizens and neighbors in this city and more distant communities where because of his great friendliness he has been beloved.

If thoughtfully we bow our heads
And close our eyes
We readily can visualize his written name,
And easily, in fancy we can see
His genial visage
And expression animate.
Still, in imagination, we can hear his chuckle,
Or the cadence of his spoken or his singing voice,
We feel his youthful spirit
For he still seems young.
He does not look plus three score years and ten,
His eyes atwinkle,
And in many ways a youth,
Although in mental attributes
He really was a sage.

Let us borrow a quatrain from our beloved poet, Dr. Oliver Wendell Holmes, written in memory of our neighbor, Whittier, the great poet of this Merrimack Valley, and author of those sacred treasures, "The Eternal Goodness" and "At Last."

"Death reaches not a spirit such as thine,
It can but steal the robe that hid thy wings,
Though thy warm breathing presence we resign
Still in our hearts its living semblance clings."

From the pen of Dr. Holmes' good friend Dr. S. Weir Mitchell, another author and poet who has shed lustre on the medical profession, we may appropriately read his

"VESPERAL"

"I know the night is near at hand,
The mists lie low on hill and bay,
The Autumn sheaves are dewless, dry;
But I have had my day.

"Yes I have had dear Lord the day
When at thy call I have the night,
Brief be the twilight as I pass
From light to dark from dark to light."

Respectfully submitted,

JOHN H. NICHOLS,
ARCHIBALD R. GARDNER,
HOWARD W. JEWETT,
Committee on Resolutions.

NOTICES

CLINIC AT THE PETER BENT BRIGHAM HOSPITAL

At 3:30 P.M. on Thursday, February 21, in the Amphitheatre of the Peter Bent Brigham Hospital, Dr. Henry A. Christian, Physician-in-Chief, Hersey Professor of the Theory and Practice of Physic in the Harvard Medical School, will give a medical clinic. To it are cordially invited practitioners and medical students. These clinics will be repeated on Thursdays until May.

On Saturdays in the wards of the Peter Bent Brigham Hospital, from 10 to 12, staff rounds will be conducted by Dr. Christian. These are open to all physicians.

A PRIZE OF FIFTY DOLLARS FOR CASE REPORTS BY INTERNS IN MASSACHUSETTS HOSPITALS

The attention of interns in Massachusetts hospitals is called to the fact that a prize of $50.00 has been offered by the Massachusetts Medical Society for the best written and most comprehensive case report, which may be submitted by one of their number holding any of the rotating internships for the year 1934-1935 in any Massachusetts hospital which is approved for intern training by the American Medical Association.

This report is to be typewritten, and when completed is to be sealed, unsigned, in a plain envelope, which in turn is to be placed together with a separate slip bearing the name and address of the contestant in a larger envelope, and send to

The Massachusetts Medical Society,
Committee on Medical Education
and Medical Diplomas,
8 Fenway,
Boston, Mass.

The contest this year closes May 1, 1935. Reports may be submitted at any time prior to that date.

AN INVITATION TO FELLOWS OF THE MASSACHUSETTS MEDICAL SOCIETY

HARVARD UNIVERSITY MEDICAL SCHOOL COURSES FOR GRADUATES

A list of activities in the Department of Pediatrics of the Children's Hospital and of the Massachusetts General Hospital, to which members of the Massachusetts Medical Society are cordially invited, appears below. These exercises are offered without fees as a part of the Courses for Graduates, of the Harvard Medical School, to those who are interested in keeping in touch with Clinical Pediatrics, without enrolling in the prescribed courses.

The Children's Hospital and the Infant's Hospital:

Clinical-Pathological Conference — Thursdays, 12:00 M. (Amphitheatre).

Clinic—Medical, Surgical and Orthopedic Services—The first Monday in each month, 4:00 P.M. (Amphitheatre).

Clinic — Alternating Rounds between Surgical Service, Peter Bent Brigham Hospital (Amphitheatre) and Surgical and Orthopedic Services, Children's Hospital (Amphitheatre) —Thursdays, 4:30 P.M.

The Massachusetts General Hospital—The Children's Medical Service:

Clinical meeting of the staff—Alternate Fridays, 12:00-1:00 P.M. (Ether Dome).

Ward Visit — Tuesdays, 2:30-4:00 P.M. (Massachusetts Eye and Ear Infirmary).

Seminar for discussion of recent investigations and literature — Tuesdays, 4:00-5:00 P.M. (Pediatric Laboratory).

MAYNARD LADD, M.D.,
In charge of Courses for Graduates,
Department of Pediatrics.

NOTICES OF MEETINGS

HARVARD MEDICAL SOCIETY

The next meeting of the Harvard Medical Society will be held in the Peter Bent Brigham Hospital Amphitheatre (Van Dyke Street entrance) Tuesday evening, February 26, at 8:15 P.M.

PROGRAM

Presentation of Cases.

Your Profession and Society. By Dr. John A. Hartwell, Professor of Clinical Surgery, Cornell University Medical School, former President of New York Academy of Medicine.

MARSHALL N. FULTON, M.D., *Secretary.*

NEW ENGLAND OPHTHALMOLOGICAL SOCIETY

The next meeting of the New England Ophthalmological Society will be held on Tuesday, February 19, 1935, at the Massachusetts Eye and Ear Infirmary, 243 Charles Street, Boston.

PROGRAM

9:30 A.M. Clinic and operating room.

11:30 A.M. Neuro-ophthalmological conference.

4:00 P.M. Presentation of cases. Clinico-pathological conference.

EVENING PROGRAM

1. Cases.
2. Paper:

Diagnosis and Treatment of Vertical Strabismus. Dr. James Watson White, New York.

BENJAMIN SACHS, M.D., *Secretary.*

NEW ENGLAND PHYSICAL THERAPY SOCIETY

The next meeting will be held at the Evans Auditorium, 82 East Concord Street, Boston, at 8 P.M. on February 20, 1935.

PROGRAM

The Present Status of Radium and X-rays in the Treatment of Malignant Disease. Frederick William O'Brien, M.D., Boston.

A motion film of a modern x-ray therapy set-up will be shown.

Discussion will be opened by Herman A. Osgood, M.D., of Boston.

Physicians and medical students are cordially invited.

The Council will meet at 7:45 P.M.

ARTHUR H. RING, M.D., *Secretary.*

Arlington.

MASSACHUSETTS PSYCHIATRIC SOCIETY

The next meeting of the Massachusetts Psychiatric Society will be held at the Boston Psychopathic Hospital on Wednesday, February 27, 1935, at 8 P.M. The following program will be presented, furnished by Dr. Clarence A. Bonner, Danvers State Hospital, and his associates:

"Review of the Problems of Bacillary Dysentery," by Dr. Salomon Gagnon.

"Sodium Fluorid Poisoning," by Dr. Leo Maletz.

"Paget's Disease," by Dr. Paul Tivnan.

OSCAR J. RAEDER, M.D., *Secretary.*

THE NORFOLK DISTRICT MEDICAL SOCIETY

A regular meeting of the Society will be held in the Hotel Kenmore, Tuesday evening, February 26, 1935, at 8:15 P.M. Telephone Kenmore 2770.

Business:

Communications:

The Use of Amniotic Fluid in Abdominal Surgery. Dr. Herbert L. Johnson.

Discussion of this paper will be opened by others prominent in this branch of surgery.

Collation.

FRANK S. CRUICKSHANK, M.D., *Secretary.*

BOSTON MEDICAL HISTORY CLUB

8 FENWAY

Monday, February 18, 1935, at 8:15 P.M.

"The Enigma of Michael Servetus and the Pulmonary Circulation." Lincoln Davis, M.D.

Illustrated by the stereopticon.

JAMES F. BALLARD, *Secretary.*

NEW ENGLAND HEART ASSOCIATION

The next meeting of the New England Heart Association will be held at the Children's Hospital, Boston, Mass., Monday, February 25, 1935, at 8:15 P.M., with the following program:

1. Two Cases of Diphtheritic Myocarditis with Recovery. Dr. Edward F. Bland.
2. Classifying 100 Living Cases of Congenital Heart Malformation. Dr. Paul W. Emerson and Dr. Hyman Green.

3. A Case of Rheumatic Heart Disease with Recovery. Dr. Maurice T. Briggs.

JAMES M. FAULKNER, M.D., *Secretary.*

SOCIETY MEETINGS, CONGRESSES AND CONFERENCES

CALENDAR OF BOSTON DISTRICT FOR THE WEEK BEGINNING MONDAY, FEBRUARY 18, 1935

Monday, February 18—

7:15 P.M. Lecture "Self-Measurement as a Group Guidance." Dr. Allen. 29 Exeter Street, Boston. Single lecture $1.50. Sponsor, Boston University School of Education.

8:15 P. M. Boston Medical History Club. Boston Medical Library, 8 Fenway.

Tuesday, February 19—

New England Ophthalmological Society. All-day session. See page 321.

12 M. South End Medical Club will meet at the Headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston.

12:30 P.M. Annual Meeting Florence Crittenton League. Speaker, Henrietta Additon, Crime Prevention Bureau, New York. Hotel Kenmore. Luncheon $1.00—Call Laf. 0160 for reservations.

1:30 P.M. Radio Program—WEEI. "The Trends in Sewage Disposal." (Continued.)

†2:30-4 P.M. Ward visit, Massachusetts Eye and Ear Infirmary.

†4-5 P.M. Seminar, Pediatric Laboratory, Massachusetts General Hospital.

4:30 P.M. Radio Program—WBZ. "Bright's Disease."

Wednesday, February 20—

8 P.M. New England Physical Therapy Society, Evans Auditorium, 82 East Concord Street, Boston.

8 P.M. Robert Breck Brigham Hospital, Clinical Meeting, 125 Parker Hill Avenue.

Thursday, February 21—

*8:30 A.M. Lecture and Clinic on Heart Disease by Dr. Christian. Peter Bent Brigham Hospital.

*12 M. Clinico-Pathological Conference. Massachusetts General Hospital.

†12 M. Clinico-Pathological Conference. Children's Hospital.

*3:30 P.M. Medical Clinic. Dr. Christian. Peter Bent Brigham Hospital.

†4:30 P.M. Surgical Clinic. Peter Bent Brigham Hospital.

Friday, February 22—

5 P.M. Radio Program—WEEI. "Eyes of the School Child." "Bedtime for Children."

Saturday, February 23—

*10-12. Medical Staff Rounds. Dr. Christian. Peter Bent Brigham Hospital.

Sunday, February 24—

4 P.M. Harvard University. (Medical School, Building D, Longwood Avenue, Boston.) Free lecture. "External Influences on Physical Activity," Dr. D. B. Dill.

*Open to the medical profession.
†Open to Fellows of the Massachusetts Medical Society.

February 15—Boston University School of Medicine Surgical Clinic at the Boston City Hospital, Cheever Amphitheatre, 12-1 P.M.

February 15—New England Roentgen Ray Society will meet at the Boston Medical Library, 8 Fenway, at 8:15 P.M.

February 18—Boston Medical History Club. See page 321.

February 19—New England Ophthalmological Society. See page 321.

February 19—South End Medical Club will meet at 12 noon at the Headquarters of the Boston Tuberculosis Association, 554 Columbus Avenue, Boston.

February 20—New England Physical Therapy Society. See page 321.

February 20—Robert Breck Brigham Hospital, Clinical Meeting will be held at the Hospital, 125 Parker Hill Avenue, at 8 P.M.

February 20—Brookfield Medical Club will meet at the Hampshire House, Ware, Mass.

February 21—Clinic at the Peter Bent Brigham Hospital. See page 320.

February 25—New England Heart Association. See page 321.

February 26—Harvard Medical Society. See page 321.

February 27—Massachusetts Psychiatric Society. See page 321.

March-May—International Medical Postgraduate Courses in Berlin. Programs and further particulars are obtainable from the Berlin Academy for Medical Postgraduate Training, Berlin NW7, Robert Koch-Platz 7 (Kaiserin Friedrich-Haus).

March 8—William Harvey Society. Dr. Percy S. Pelouze, University of Pennsylvania, will speak on "Neisseriana."

March 11, 12, 13—Surgeons to meet in Jacksonville, Florida (Southeastern Surgical Congress). See page 83, issue of January 10.

MASSACHUSETTS DIETETIC ASSOCIATION

March 12—Tuesday, 8 P.M. "The Effect of Diet on Anemia," Dr. Lewis Diamond, Instructor in Medicine, Harvard University Medical School, Associate Physician, Children's Hospital.

March 19—Tuesday, 2 P.M. Field Trip: Visit Storehouse, First National Stores.

April 9—Tuesday, 8 P.M. "Small Hospital Problems," Miss Margaret Copeland, Superintendent, Free Hospital for Women.

April 29 - May 3, 1935—The American College of Physicians will meet at Philadelphia. For information address Mr. E. R. Loveland, Executive Secretary, 133-135 South 36th Street, Philadelphia, Pa.

June, 1935—Medical Library Association will meet in Rochester, N. Y. For details address the Secretary: Miss Frances N. A. Whitman, Librarian, Harvard University Schools of Medicine and Public Health, Boston, Mass.

June 27-29 inc.—British National Association for the Prevention of Tuberculosis will be held at Southport, England. Persons desiring further information should write to Miss F. Stickland, Secretary of the Association at Tavistock House North, Tavistock Square, London, W. C. I., England.

July 22-27—Seventh International Congress on Industrial Accidents and Diseases, Brussels, Belgium. The American Committee of the Congress is under the chairmanship of Dr. Fred H. Albee, New York, for the Section on Accidents, and that of Dr. Emery R. Hayhurst, Columbus, Ohio, for Industrial Diseases. Physicians interested in the Congress or in the medical tour in conjunction with it may address the Secretary, Dr. Richard Kovacs, 1100 Park Avenue, New York City.

DISTRICT MEDICAL SOCIETIES

ESSEX NORTH DISTRICT MEDICAL SOCIETY

The Annual Meeting will be held in May. Time, place and subject to be announced.

E. S. BAGNALL, M.D., Secretary.

FRANKLIN DISTRICT MEDICAL SOCIETY

Meetings will be held on the second Tuesday of March and May at the Weldon Hotel, Greenfield, Mass.

CHARLES MOLINE, M.D., Secretary.
Sunderland.

MIDDLESEX EAST DISTRICT MEDICAL SOCIETY

March 13—Wakefield.
May 8—Winchester.

K. L. MACLACHLAN, M.D., Secretary.
1 Bellevue Street, Melrose.

NORFOLK DISTRICT MEDICAL SOCIETY

February 26—See page 321.

March 26—Fernald School for Feeble-Minded, Waverley. Details to be announced.

May—Annual Meeting. Date, time and place to be announced.

PLYMOUTH DISTRICT MEDICAL SOCIETY

March—Plymouth County Hospital.
April—Lakeville Sanatorium.

SUFFOLK DISTRICT MEDICAL SOCIETY

March 27—Clinical Meeting at the Boston Lying-In Hospital.

April 24—Clinical Meeting at the Children's Hospital.

The medical profession is cordially invited to attend these meetings.

ROBERT L. DeNORMANDIE, M.D., President.
GEORGE P. REYNOLDS, M.D., Secretary.

WORCESTER DISTRICT MEDICAL SOCIETY

March 13—Wednesday evening. The Memorial Hospital, Worcester, Mass. 6:30 P.M.: Buffet supper. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Buffet supper complimentary by the Hospital.

April 10—Wednesday evening. Worcester Hahnemann Hospital, Worcester, Mass. 6:30 P.M. Dinner. 7:30 P.M.: Scientific program and business session. Announcement of subjects and speakers to be presented at a later date. Dinner complimentary by the Hospital.

May 8—Wednesday afternoon and evening. Annual Meeting of the Worcester District Medical Society. The time and place of this meeting will be announced later.

ERWIN C. MILLER, M.D., Secretary.
27 Elm Street, Worcester.